STUDY ON ASSOCIATION OF MATERNAL PERIODONTITIS AND PREECLAMPSIA

Dissertation submitted to

THE TAMILNADU

Dr. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirement

For the award of

M.S.DEGREE- BRANCH – II

OBSTETRICS AND GYNAECOLOGY

THE TAMIL NADU Dr. M.G.R MEDICAL UNIVERSITY

INSTITUTE OF OBSTRETICS AND GYNAECOLOGY

MADRAS MEDICAL COLLEGE, CHENNAI.

MAY - 2018

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"STUDY ON ASSOCIATION OF MATERNAL PERIODONTITS AND PREECLAMPSIA"** is the bonafide original work done by **Dr.SURYAKIRANMAYI.R** under the guidance of **Dr.SARALA, MD OG,** Professor of Obstetrics and Gynecology, IOG, Chennai in partial fulfillment of the requirements for MS Obstetrics and Gynecology branch II examination of the Tamil Nadu Dr.MGR Medical university to be held in MAY 2018 The period of post graduate study and training from **MAY 2015 TO MAY 2018**

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DECLARATION

I solemnly declare that this dissertation **"STUDY ON ASSOCIATION OF MATERNAL PERIODONTITS AND PREECLAMPSIA"** was prepared by me at Institute of obstetrics and gynaecology, Chennai, under the guidance and supervision of **Dr. SARALA MD, DGO,** Professor of Obstetrics and Gynaecology, Institute of obstetrics Chennai.

This dissertation is submitted to **The Tamil nadu Dr. M.G.R.Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.S. (Obstetrics and Gynaecology).**

Place: Chennai

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ACKNOWLEDGEMENT

I gratefully acknowledge and sincerely thank **Dr.Narayana Babu M.D., DCH.,** Dean, Madras Medical College and Research Institute, Chennai for allowing me to use the facilities and clinical materials available in the hospital.

My sincere thanks and gratitude to **Dr.D.Tamil selvi, M.D.,D.G.O.,** Director and Superintendent, Institute of Social Obstetrics, for granting me permission to utilize the facilities of the institute for my study.

I am extremely grateful to our Professor, **Dr. SARALA.J. M.D., D.G.O.,** Institute of Obstetrics and Gynaecology and Government Women and Children hospital, Egmore, Chennai for her valuable guidance, motivation, and encouragement given during the study.

I humbly thank all the Professors and Assistant Professors of IOG, Egmore and Government Kasturba Gandhi Hospital, Triplicane for all their help during the course of the study My sincere thanks to **Mr.Ashok kumar**, Statistician for helping me in analysing the results of my study.

My special thanks to my family and friends for their physical help and moral support without which nothing would have been possible.

I am immensely grateful to all the patients who took part in the study.

CONTENTS

INTRODUCTION

“Mouth is the mirror of the whole body” is an age-old concept with its relevance even today. Periodontitis, otherwise known as pyorrhea, is a set of inflammatory diseases affecting the tissues surrounding the teeth. It involves progressive loss of the alveolar bone around the teeth, and if left untreated, can lead to the loosening and subsequent loss of teeth.It is caused by microorganisms that adhere to and grow on the tooth's surfaces, along with over-aggressive immune response against these microorganisms. Diagnosis of periodontitis is established by inspecting the soft tissue around the teeth with a probe to determine the amount of bone loss around the teeth.

Periodontal diseases

Periodontal diseases are plaque induced infections and pathological changes of the periodontium, divided into two categories: gingivitis and periodontitis. Gingivitis, the mildest form of periodontal diseases, affects up to 90% of the population and manifests clinically as edema and erythema of the marginal gingiva.

Gingivitis does not affect the underlying tooth anchoring structures (connective tissue and alveolar bone) and is readily reversible by effective tooth cleaning and oral hygiene home care. Inflammation that extends deeper into the supportive tissues and involves the periodontal ligament and alveolar bone is known as periodontitis. This apical extension of the inflammatory process results in progressive destruction of tooth anchoring collagen fibers and

supportive bone structures and can clinically lead to the formation of deepened soft tissue pockets between the gingiva and the surface of the tooth. Once periodontal pockets are formed and colonised with bacteria, improving oral hygiene alone is no longer sufficient to reverse the inflammatory process. Intensive professional periodontal treatment, including supra- and subgingival scaling and rootplaning (SRP) and surgical pocket elimination is necessary to restore periodontal health.

Micro -organisms involved in periodontal diseases

A shift in microbial species in the dental plaque from a relatively harmless gram-positive,facultative anaerobic fermentative population to a predominantly gram-negative,anaerobic and proteolytic virulent population has been strongly associated with periodontal tissue breakdown. Although more than 500 bacterial species have been identified in the dental plaque only a small proportion of these micro-organisms are considered to play a role in the development of periodontitis. Key pathogens associated with periodontitis in adult subjects are the gram-negative micro-organisms and the gram-positive *Parvimonas micra (P. micra)*. These bacteria produce a variety of toxins, such as outer membrane lipopolysaccharides, outer membrane lipids or lipoproteins, peptidoglycan and proteolytic enzymes, which may have detrimental effects on the periodontal tissues.Especially *P. gingivalis*, a black pigmented anaerobic micro-organism, has been reported to possess a variety of virulence factors, including

lipopolysaccharide (LPS) *P.gingivalis* LPS has the capacity to upregulate the host's immune response, which may lead to an increased production of local inflammatory mediators and effector molecules including cytokines, chemokines and matrix metalloproteinases, and finally to periodontal breakdown.

In addition, periodontitis also has a genetic component. Various single-nucleotide polymorphisms (SNPs), for example in the IL-1β, IL-1RN and TLR4 genes, have been associated with chronic and aggressive periodontitis. Thus, not only the mere presence of pathogenic micro-organisms in the biofilm, but also the genetically determined mode of host inflammatory responses to these pathogens determines the clinical manifestations of periodontitis, e.g. the severity of periodontal tissue breakdown. It has indeed been shown that not all individuals are equally affected by the accumulation of dental plaque. Some susceptible individuals may develop aggressive forms of periodontitis at a young age, while others never, or very slowly develop periodontitis. A significant portion of the host predisposition to periodontitis may therefore relate to excessive innate host defenses and the subsequent activation of the inflammatory response

Periodontal disease is a multifactorial, chronic, gram-negative infection associated with atherosclerotic thromboembolic events. It may act as a chronic reservoir of endotoxins and inflammatory cytokines that initiate and exaggerate atherogenesis and thrombogenesis. Pregnancy-induced gingivitis and periodontitis are two oral conditions with prevalence rates ranging from 30 to 100% and from 5 to 20%, respectively.

Preeclampsia

The second most leading cause for maternal mortality is hypertensive disorder of pregnancy, which accounts for 15% of total deaths and still births. Preeclampsia is a complex disorder affecting about 5–10% of the obstetric population, resulting from deficient placental implantation during the first half of pregnancy and hypertensive disorders after 20^{th} week of gestation in a previously normotensive woman.

Pathogenesis of preeclampsia

Preeclampsia is a maternal multi-organ disease, clinically manifest during the second half of pregnancy by hypertension (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) with proteinuria (≥300 mg/24 hours or 2+ dipstick), often accompanied with varying dysfunction of major organs as the liver, the kidneys and the brain. It is one of the leading causes of maternal and foetal morbidity and mortality. Several factors have been implicated in the pathogenesis of preeclampsia, including genetic, immunologic,

inflammatory, ethnic, socioeconomic and environmental factors, but til now the exact cause and pathogenesis are not fully clarified. The current leading hypothesis is that the pathological processes that underlie preeclampsia occur in two stages. Stage one starts with abnormal placentation and impaired vascular remodeling of the myometral spiral arteries which supply the placental bed. This impaired remodeling is most likely the result of deficient trophoblast invasion into the myometrial arteries. The consequence is insufficient placental perfusion in the second half of pregnancy, when the increasing demands of the growing fetus can no longer be met and leading to placental oxidative stress and the release of various bioactive factors into the maternal circulation. These bioactive factors include inflammatory cytokines such as TNF-α andIL-6, as well as syncytiotrophoblast membrane microparticles (STBMs), which may challenge maternal inflammatory cells. They also include antiangiogenic factors such as soluble endoglin (sEng), the soluble form of the vascular endothelial growth factor (VEGF) receptor (sFlt-1) and placental growth factor (PlGF), which challenge the maternal vascular endothelium. This leads to stage two, endothelial dysfunction and an excessive inflammatory response leading to the maternal clinical features of the syndrome. Preeclampsia, therefore, is most likely the result of a generalised exacerbation of the inflammatory response, including activation of inflammatory cells and endothelial cells

Preeclampsia and periodontitis have been found to be associated with high circulating levels of tumor necrosis factor-alpha (TNF-α), interleukin (IL)-10, and IL-6 resulting in inflammatory vascular damage leading to placental endothelial alterations. Periodontal microbiota plays a significant role in systemic diseases directly through a pro-inflammatory effect or indirectly through the host-mediated destruction. The similarities in their pathophysiologies have led to the hypothesis of periodontal disease being a risk factor for preeclampsia.

AIM OF THE STUDY

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The aim of the present study was to evaluate the association between maternal periodontitis and preeclampsia.

Also the association of periodontitis and preeclampsia after matching for primi Parity.

REVIEW OF LITERATURE

Periodontitis

Periodontal disease is a disease, or more likely a number of diseases of the periodontal tissues that results in attachment loss and destruction of alveolar bone. The natural history of periodontal disease, in some but not all patients, results in tooth loss. Periodontal disease, however, encompasses a wider spectrum of diseases than just periodontitis and the recognition of these diseases requires a diagnosis be made.

Diagnosis is the recognition of the presence of a disease. Clinical diagnosis of periodontal disease is made by the recognition of various signs and symptoms in the periodontal tissues which herald a departure from health. The diagnosis of periodontal disease demands a firm knowledge of what constitutes periodontal health. The healthy periodontium,of which only the gingival tissues may be directly observed, is described as being stippled, pale pink or coral pink, in the Caucasian , with various degrees of pigmentation in other races. It is tightly adapted to the underlying tissues, with a knife edge margin where it abuts the tooth, The gingival margin is located, in the absence of pathology, at the cemento-enamel junction. It displays a scalloped edge configuration highest interdentally, where it constitutes the interdental papilla and lowest buccally and lingually. There is a gingival crevice where it abuts the tooth which in health is 1–3 mm deep.

There is an absence of bleeding from the crevice on gentle probing. The crevice in health will show a small amount of interstitial fluid, gingival crevicular fluid. The lateral wall of the crevice constitutes the free gingival margin. From the most apical extent of the free gingival to the mucogingival junction is the attached gingival which varies in width from 1 to 9 mm and has a stippled surface. It is an immobile tissue tightly bound down to the bone as a mucoperiostium and is a keratinized mucosa well suited to resist injury. Apical from the mucogingival junction and continuous with the lining mucosa of the mouth is the alveolar mucosa, which is freely mobile and surmounted by a non-keratinized epithelium. It is generally thought that alveolar mucosa functions poorly as a marginal tissue and areas where there is lack of attached gingival may constitute mucogingival problems. Departures from this concept of the healthy periodontium may suggest the presence of disease.

Key pathogens associated with periodontitis in adult subjects are the gram-negative micro-organisms *Aggregatibacter actinomycetemcomitans (A.actinomycetemcomitans), Porphyromonas gingivalis (P. gingivalis),* Prevotella intermedia (P. intermedia), Tannerella forsythia (T. forsythia), *Fusobacterium nucleatum* (*F. nucleatum*) and the gram-positive *Parvimonas micra (P. micra)*.

These bacteria produce a variety of toxin, such as outer membrane lipopolysaccharides, outer membrane lipids or lipoproteins, peptidoglycan and proteolytic enzymes, which may have detrimental effects on the periodontal tissues

Host-parasite interactions

One of the first ways in which the host's immune system recognises different microorganisms is through pattern recognition receptors (PRR) such as Toll-like receptors(TLRs), RIG-I-like receptors (RLRs), NOD-like receptors (NLRs) and DNA receptors(cytosolic sensors for DNA) on innate immune cells. TLRs are specific pattern recognition receptors present on a variety of cells, such as monocytes, macrophages and dendritic cells, as well as on (oral) epithelial cells, gingival fibroblasts and endothelial cells. These TLRs detect so-called damage-associated molecular patterns(DAMPs). A subtype of DAMPs, pathogen-associated molecular patterns (PAMPs),arise from pathogens and alarm an individual to invading pathogens. Several surface components present on micro-organisms, like LPS, the major component of gram-negative bacterial cell walls, or peptidoglycan, the major component of grampositive bacterial cell outer membranes are PAMPs and are recognised by TLRs.

Ligation of TLRs by LPS or peptidoglycan results in activation of the intracellular signalling pathway and ultimately in the production of an array of cytokines. These cytokines modulate inflammatory reactions. At present, 12 mammalian TLRs have been identified, of which TLR4 and TLR2 have been studied most frequently. TLR4 plays an important role as a receptor for bacterial LPS from gram-negative micro-organisms, while TLR2 mainly recognises peptidoglycan or lipoproteins/lipopeptides from gram positive bacteria. Activation of TLR4 leads to the production of pro-inflammatory cytokines, like tumour necrosis factor alpha (TNF-α), interleukin-1 (IL-1), IL-6, IL-12. Activation of TLR2 leads to the production of prostaglandin E2 (PGE2), IL-10 and IL-13, of which in particular IL-10 has been recognised as a potent immunosuppressive and anti-inflammatory molecule. Thus, the micro-organisms in the dental plaque,that consists of an array of gram-positive and gram-negative species, may influence the nature of the host response to this polybacterial challenge, by activating specific TLRs.

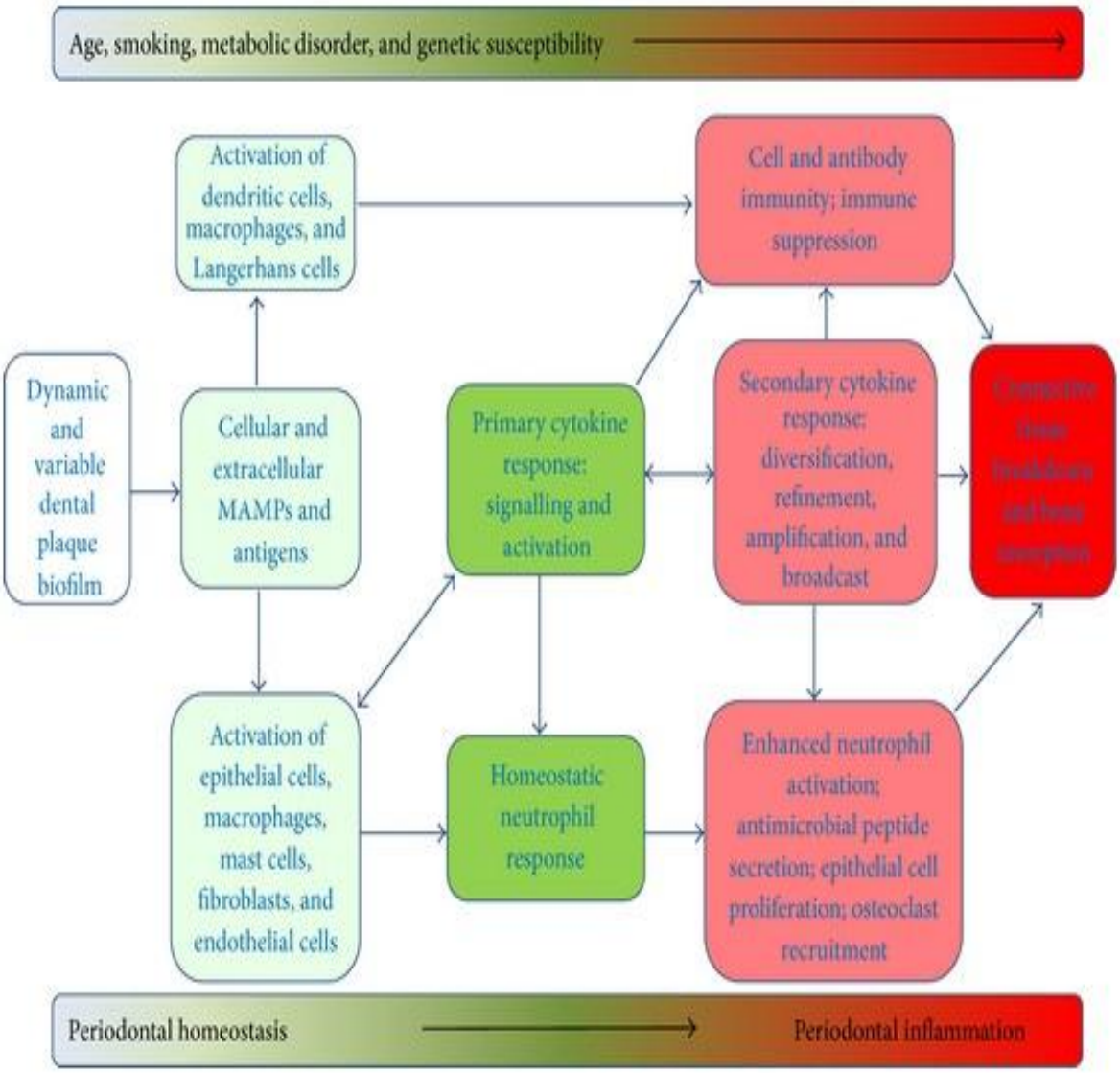
In addition, periodontitis also has a genetic component. Various single-nucleotide polymorphisms (SNPs), for example in the IL-1β, IL-1RN and TLR4 genes, have been associated with chronic and aggressive periodontitis. Thus, not only the mere presence of pathogenic micro-organisms in the biofilm, but also the genetically determined mode of host inflammatory responses to these

pathogens determines the clinical manifestations of periodontitis, e.g. the severity of periodontal tissue breakdown. It has indeed been shown that not all individuals are equally affected by the accumulation of dental plaque. Some susceptible individuals may develop aggressive forms of periodontitis at a young age, while others never, or very slowly develop periodontitis.

The findings that increased levels of inflammatory mediators, such as TNF-α, IL-1β and PGE2, correlate with periodontal destruction and the fact that these mediators aggravate the inflammatory response, has led to the hypothesis that some individuals may be "high-responders" and respond to periodontal infection with high levels of inflammatory mediators which results in periodontal breakdown. Indeed, increased TNF-α and PGE2 production has been observed in patients with aggressive (early-onset) periodontitis as compared with patients with generalised periodontitis. A significant portion of the host predisposition to periodontitis may therefore relate to excessive innate host defenses and the subsequent activation of the inflammatory response.

Physiology of normal pregnancy

The placenta which is totally derived from the foetus after conception invades and grows totally supported by the maternal uterine tissue. As the foetus grows, the need for the nutrition increases causing decrease in space which becomes a critical parameter for the survival of both the mother and the foetus. As pregnancy progresses amniotic fluid levels of prostaglandin E2 (PG E2) and inflammatory cytokines such as TNF-α and IL-1β rise until a critical threshold level is reached to induce rupture of the amniotic sac membrane, uterine contraction, cervical dilation and delivery.


Age, smoking, metabolic disorder, and genetic susceptibility
Activation of dendritic cells, macrophages, and Langerhans cells
Cell and antibody immunity; immune suppression
Dynamic and variable dental plaque biofilm
Cellular and extracellular MAMPs and antigens
Primary cytokine response: signalling and activation
Secondary cytokine response: diversification, refinement, amplification, and broadcast
Activation of epithelial cells, macrophages, mast cells, fibroblasts, and endothelial cells
Homeostatic neutrophil response
Enhanced neutrophil activation; antimicrobial peptide secretion; epithelial cell proliferation; osteoclast recruitment
Periodontal homeostasis
Periodontal inflammation

Periodontal diseases during pregnancy

Periodontal health during pregnancy has engendered much interest over the past years. For many years, it has been established that during pregnancy (in 30-100% of women), a progressive increase in gingival inflammation is observed. The phenomenon referred to as pregnancy gingivitis and disappears post-partum with no permanent effects on the periodontal attachment. Although the exact mechanisms remains unknown, it is considered that elevated levels of circulating progesterone or estrogen during pregnancy contribute to enhanced gingival vascular permeability and enhanced gingival exudates. This may lead to clinical alterations of the gingiva, such as increased redness, oedema and a greater bleeding tendency, which clinically resemble inflammation. Unfortunately, this condition not only leads to greater gingival probing depths, but subsequently may transform the gingiva into an environment that may favour the overgrowth of specific bacteria. In a recently conducted cohort study, the presence of *P. gingivalis* significantly contributed to the worsening of gingival inflammation during the second and third trimester of pregnancy. Gingival lesions are classified into two broad categories. Plaque induced and non-plaque induced. Dental plaque induced lesions (gingivitis) may be purely plaque related with or without local contributing factors or may be modified by systemic factors, medications or by malnutrition. It should be noted that, although by definition gingivitis has been traditionally described as being associated with a periodontium where there has been no loss of attachment, it is

possible for gingivitis to occur on a periodontium with a reduced attachment level which is stable and not experiencing progressive loss of attachment. Non-plaque induced gingival lesions encompass those caused by specific bacterial, fungal or viral infections, genetic origin, systemic conditions (dermatological conditions, allergic reactions), foreign body reactions, trauma lesions and a catch all, not otherwise specified, for forms of gingivitis that do not fit neatly into any of the other areas. The dermatological disease, lichen planus which is common, may occur in the mouth without skin lesions and is frequently confused and misdiagnosed as plaque induced gingivitis.
Pregnancy gingivitis may also be related to altered immune and inflammatory responses during pregnancy. It is evident that during normal pregnancy the inflammatory response is mildly activated. This activation of the inflammatory response during normal pregnancy is characterised by increased expression of activation markers on monocytes and granulocytes, differences in monocyte cytokine production and increased plasma levels of inflammatory markers, such as CRP.

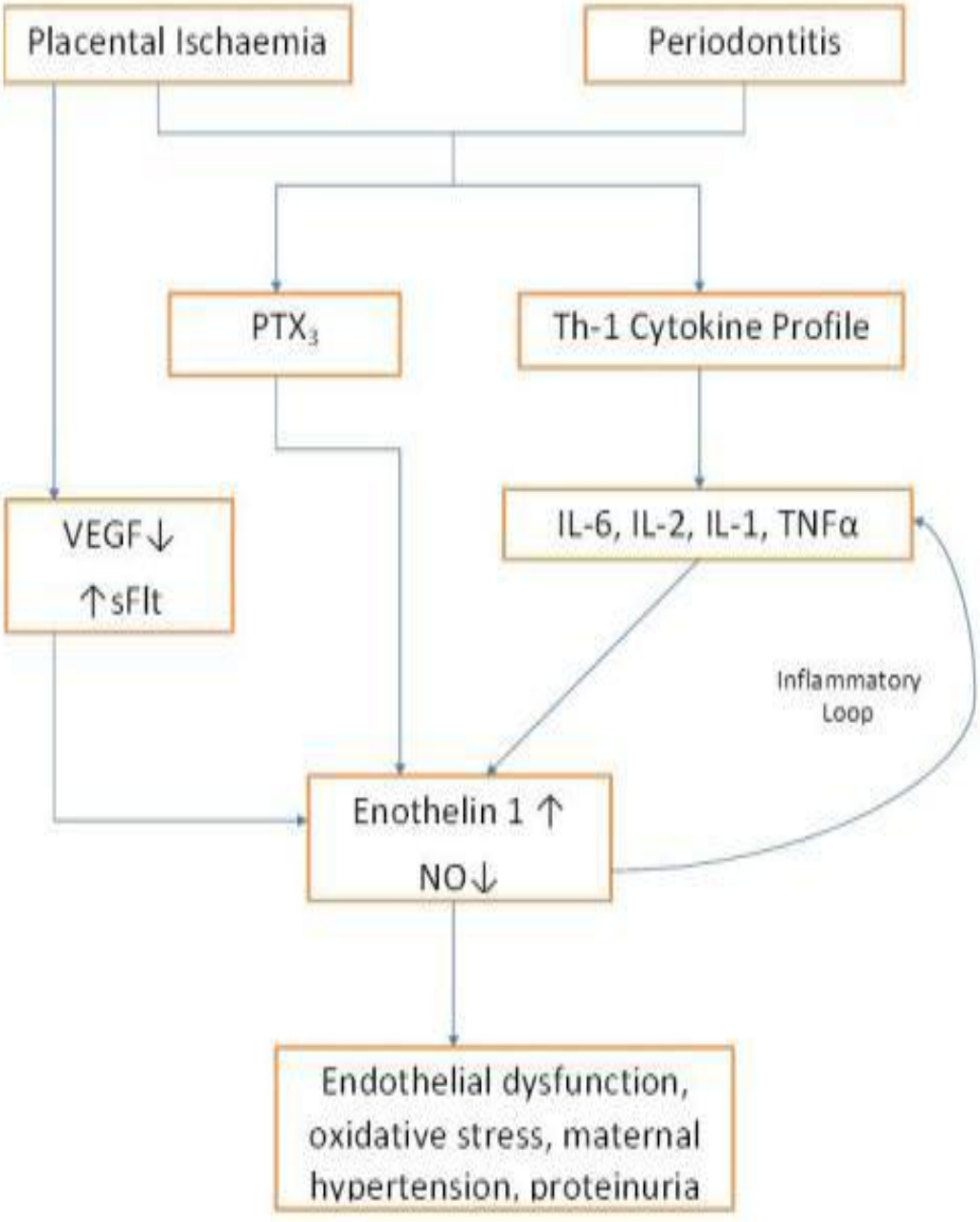
This led to the concept that pregnancy is a pro-inflammatory condition and might partly explain the increased susceptibility to gingivitis during pregnancy.

In the year 1996, Offenbacher et al. were among the first who reported the relationship between periodontal disease on one hand, and premature labour and low birth weight on the other . As a result, researches were carried out on grounds of finding the link between periodontal disease and the complications of pregnancy such as preeclampsia. Preeclampsia is a rapidly progressing syndrome in pregnant women that is characterized by the blood pressure equal or greater than 140/90 mmHg after the week 20 of pregnancy along with +1 proteinuria in urine sample.

Preeclampsia is one of the main causes of maternal mortality and the death of the fetus . The manifestations are increased blood pressure, maternal protein urea and also associated with high levels of pro inflammatory cytokines. Increased maternal serum levels of pro inflammatory cytokines such as IL-1, IL-8, IL-6 are also associated with pre-maturity or low birth weight. C-reactive protein which is an acute phase reactant which is synthesized in response to pro-inflammatory cytokines by the liver is also associated with pre-eclampsia.Where CRP is a short pentraxin produced in the liver, another counterpart in the pentraxin family is the long pentraxin called Pentraxin 3(PTX3). PTX3 is produced by a variety of cells including fibroblasts, mononuclear phagocytes, vascular endothelial cells and smooth muscle cells. Pentraxin 3 is known to cause endothelial dysfunction and inflammation in preeclamptic women. PTX3 levels have been found to be elevated at the time of normal pregnancy.

However, the levels are significantly higher in preeclamptic women when compared to normal pregnancy. Other factors like Endothelin 1, Sflt-1, Nitric Oxide, Tumour Necrosis Factor alpha, Thromboxane A2 have also been suspected to play a role in Preeclampsia. The reasons of the syndrome could be listed as abnormal thromboplastic invasion of uterine vessels, immunologic mismatch between mother and child, lack of mother's proper compatibility with the cardiovascular and inflammatory changes of pregnancy, diet, and genetics .

Figure 1: PATHOGENESIS OF PREECLAMPSIA



While it has been traditionally assumed that preeclampsia is a self-limited disease that resolves once the baby and placenta are delivered, some studies have shown that this maternal endothelial dysfunction can last for years after the episode of preeclampsia .The children born after a pregnancy complicated by preeclampsia have also been shown to be at high risk for complications like diabetes mellitus, cardiovascular disease, and hypertension Moreover, it is believed that paternal genes also play an important role in the development of preeclampsia. This is evidenced by the risk of preeclampsia in women with pregnancies who have previously been involved in pregnancies of men , complicated with preeclampsia. A large genetic association study of preeclampsia was published by Goddard et al that reported a study evaluating 775 SNPs in 190 genes in more than 350 preeclamptic mother and offspring pairs and 600 control pairs. They detected six genes with significant maternal-fetal genotype interaction related to preeclampsia in IGF1, IL4R, IGF2R, GNB3, CSF1 and THBS4. These findings and others suggest a multifactorial polygenic inheritance with a genetic component in the development of this disease.

Periodontal diseases as a risk factor for pregnancy complications

It was first demonstrated in 1994 in an animal model that the presence of periodontal pathogens might have a negative effect on pregnancy outcome. Collins et al demonstrated that *P. gingivalis*, infused in the pregnant hamster, could elicit low birth weight110. In humans, the first evidence of a possible association between periodontitis and adverse pregnancy outcomes was published in 1996 by Offenbacher et al, reported a relationship between periodontitis and preterm low birth weight. Since then, the relationship between periodontitis and complications of pregnancy has become a topic of research . Various observational studies were carried out, and many systematic reviews support the hypothesis that periodontitis is associated with preterm labour and other conditions complicating pregnancy, such as low birth weight, stillbirth, miscarriage and fetal growth restriction. Perhaps the strongest and most convincing evidence of a relationship between periodontitis and preterm low birth weight until now comes from a metaanalysis published by Vergnes et al. The authors concluded from their data that pregnant women with periodontitis have a 2.8-fold increased risk on preterm birth.

There are several pathophysiological mechanisms by which periodontitis may interfere with pregnancy. A route via the peripheral circulation to the placenta is most likely to be involved, since periodontopathic bacteria have been found in human placental tissues, in amniotic fluid or in chorionic tissues. The presence of periodontal micro-organisms in foetal and maternal tissues has been shown to induce chorioamnionitis, an intrauterine status of inflammation in tissues of either mixed fetal-maternal or fetal origin. Chorioamnionitis is an independent risk factor for preterm birth and fetal morbidity. Secondly, the translocation of bacterial products like LPS, and the effect of maternally produced inflammatory mediators like TNF-α and PGE2 could initiate labour. As PGE2 mediates cervical ripening and stimulates uterine contractions, it plays a role in the onset of labour. It has been postulated that the increased TNF-α and PGE2 levels accompanying periodontitis may interfere with normal physiological mechanisms of parturition, resulting in preterm labour.

If periodontal infections are a cause of preterm birth, it might be expected that eradication of such infections would reduce the risk of preterm birth. Although the results of initial studies of periodontal treatment to reduce the risk of preterm labour were positive, these findings could not consistently be confirmed in later studies. However, in a recently published meta-analysis of RCTs found a significant reduction of preterm birth after periodontal treatment in a subgroup of high risk of preterm birth.

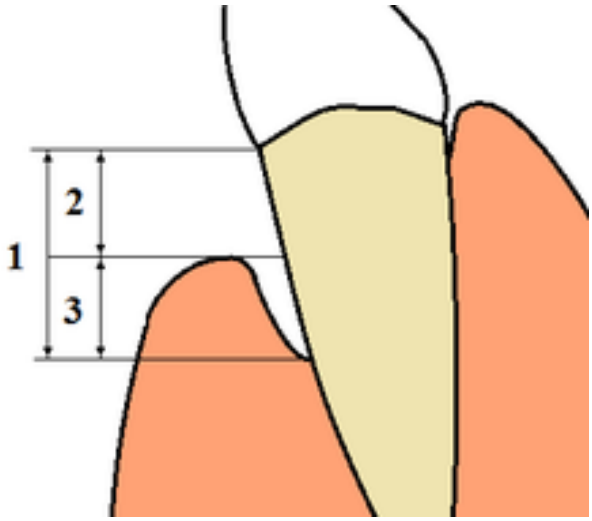
Severity of periodontal disease

The "severity" of disease refers to the amount of periodontal ligament fibers that have been lost, termed "clinical attachment loss". According to the American academy of periodontits the classification of severity is as follows:

Mild: 1–2 mm (0.039–0.079 in) of attachment loss

Moderate: 3–4 mm (0.12–0.16 in) of attachment loss

Severe: ≥ 5 mm (0.20 in) of attachment loss



1.clinical attachment loss(total loss of attachment) is the sum of

2.Gingival recession and

3.probing depth

Systemic effects of periodontal diseases

The local infection and subsequent inflammatory response of the host may not be limited to the periodontal tissues. With daily tooth brushing or even gentle mastication, periodontal micro-organisms or their bacterial products like LPS may enter the blood stream and disseminate into the systemic circulation. This chronic or even permanent bacteremia or endotoxemia may activate inflammatory cells and endothelial cells, and thus lead to endothelial cell activation and dysfunction as well as generalised inflammation, with increased numbers of peripheral blood leukocytes and increased levels of IL-6 and C-reactive protein (CRP). Most of these systemic markers of inflammation are also regarded as predictive markers for cardiovascular diseases (CVD) and it is believed that the systemic level of inflammation in periodontitis increases the risk for CVD. Indeed, over the last few years, meta-analyses of observational studies showed that patients with periodontitis are at increased risk of developing cardiovascular diseases (CVD). The persistent evidence already has led to a consensus paper on the relationship between CVD and periodontitis, which was published concurrently in two leading journals in their fields, the American Journal of Cardiology (AJC), and the Journal of Periodontology (JOP). It was confirmed in a meta-analysis by D'Aiuto et al in 2013 that periodontal treatment reduced CVD biomarkers and improves endothelial function.The importance for periodontal diagnosis and therapy in atherosclerotic individuals to improve their cardiovascular risk profile has been

further emphasised upon by Gunupati et al, who showed that periodontal treatment reduced biomarkers for atherosclerotic disease in periodontitis patients already suffering from CVD.

Following a systematic review of the association between respiratory diseases and oral health, it was concluded that there is a fair evidence of an association of pneumonia with oral health and that a good evidence exists that improved oral hygiene and frequent professional oral health care reduce the progression or occurrence of respiratory diseases among the high-risk elderly living in nursing homes, especially those in intensive care units. Scannapieco *et al*. showed that lung function decreased with increasing periodontal attachment loss. Therefore, they concluded that a potential association between periodontitis and chronic pulmonary diseases like chronic obstructive pulmonary disease (COPD) may exist. In one of the studies by Scannapieco *et al*., they found a nearly fivefold increase in chronic respiratory diseases in subjects that had poor oral hygiene when compared to those with good oral hygiene. Poor oral hygiene and periodontitis influence the incidence of pulmonary infections, especially nosocomial pneumonia episodes in high-risk subjects. The oral cavity has long been considered a potential reservoir for respiratory pathogens. The mechanism of infection could be aspiration of oral bacteria capable of causing pneumonia into the lungs, colonization of dental plaque by respiratory pathogens, followed by aspiration.

Other mechanisms include: alteration of the mucus surface by salivary enzymes in periodontitis, leading to an increase in adhesion and colonization of respiratory pathogens; destruction of salivary pellicles on pathogenic bacteria by periodontal disease-associated enzymes; and alteration of respiratory epithelium by cytokines from periodontal disease, facilitating the infection of the epithelium by respiratory pathogens.

Musculoskeletal system

People with moderate to severe periodontitis have been found to have a higher risk of suffering from rheumatoid arthritis. It has been suggested that periodontal disease could be a causal factor in the initiation and maintenance of the autoimmune inflammatory response that occurs in rheumatoid arthritis. de Pabio *et al*. stated that if this assertion is proven, chronic periodontitis might represent an important modifiable risk factor for rheumatic disease. It is thought that a remarkable similarity in the pathogenesis of periodontal diseases and rheumatoid arthritis exists. A poorly modulated inflammatory response is believed to drive both diseases, resulting in oxidative stress induced tissue injury. In addition, there has been an increasing interest in the interrelationship between systemic osteoporosis, oral bone loss, tooth loss, and risk factors for these conditions, and a positive correlation between systemic bone mass and oral bone loss had been shown.

Reproductive system

Studies have shown that there is a significant association between preterm birth and/or low birth weight and periodontitis, irrespective of parity, race, and maternal age. It has also been stated that periodontitis appears to be an independent risk factor for poor pregnancy outcome and preliminary evidence suggests that periodontal intervention may reduce this adverse pregnancy outcome.This is said to occur because bacterial infection results in the activation of cell-mediated immunity and the subsequent production of cytokines such as interleukins (IL-1, IL-6) tumor necrosis factor (TNF-a), and prostaglandins (PDE_2), which have been implicated in the mechanism of labor. The recently proposed mechanism of labor suggests that the intra-amniotic levels of these mediators rise steadily throughout pregnancy until a threshold is reached at which labor is induced. Thus, it raises the possibility that the presence of infection results in an abnormally elevated production of the normal physiological mediators of parturition, which may trigger births, also resulting in low birth weight. It is also hypothesized that sub-clinical infections such as periodontal disease contributed to premature delivery and low birth weight as a result of pathogenic microorganisms, or indeed their microbial products such as lipopolysaccharide (LPS), reaching the uterus via the blood stream, inducing cytokine release in the deciduas or in the membranes, resulting in increased prostaglandin, or indeed uterine muscle contraction. Recently, it was discovered that pregnant women with periodontal disease are more likely to develop gestational diabetes mellitus than are pregnant women with healthy gum.

Endocrine system

While it has been established that people with diabetes are more prone to developing periodontal disease, new research is suggesting that periodontal disease may, in turn, be a risk factor for diabetes. Periodontal disease can cause bacteria to enter the bloodstream and activate immune cells. These activated cells produce inflammatory biological signals (cytokines) that have a destructive effect throughout the entire body. In the pancreas, the cells responsible for insulin production can be damaged or destroyed by the chronic high levels of cytokines. Once this happens, it may induce Type 2 diabetes, even in otherwise healthy individuals with no other risk factors for diabetes. Because periodontal disease contributes to the progression of impaired glucose tolerance to diabetes mellitus and to hyperglycemia in individuals with established diabetes, proactive, preventive dental and diabetes self-care, as well as regular dental and diabetes assessment had been suggested as important management strategies.

Malignancy

Earlier literature review showed that chronic periodontitis is an independent clinical high-risk profile for head and neck squamous cell carcinoma (HNSCC), especially in the oral cavity, followed by the oropharynx and larynx. In a prospective cohort study by Michaud *et al*., a significant association was found

between the history of periodontitis and risk of developing lung, kidney, pancreas, and hematological cancers. These associations are said to persist in a number of studies, after adjustment for major risk factors, including cigarette smoking and socioeconomic status. However, the most consistent increased risk was noted in the studies of oral and esophageal cancers and periodontal disease. Gastric and pancreatic cancers had an association in most, but not all studies. Lung, hematological, and other cancers were less consistently associated or did not have sufficient studies to determine a predictable pattern. Furthermore, Tezal *et al*. reported that patients with periodontal disease were more likely to have poorly differentiated oral cavity squamous cell carcinoma (SCC) than those without periodontitis. These findings were said to have implications for practical and safe strategies for prevention, diagnosis, and treatment of HNSCC (please, check the second line above. The error in the abbreviation above was spotted and corrected). The possible link between periodontitis and malignancy is not clear, but lifetime cumulative infection exposure is being queried

Other oral health conditions impacting on systemic health

The oral cavity has a multitude of functions in relation to daily life such as: food intake, speech, social contact, and appearance. Poor oral health has thus the potential of affecting the quality of life. Apart from chronic periodontitis discussed above, there are a number of other oral health conditions that impact on systemic health. For example, the number of teeth is a significant and independent risk indicator for early mortality and poorer general health status.

Masticatory disability has likewise been related to early mortality.Oral health and nutrition have a synergistic relation. Oral infectious diseases and acute, chronic, and terminal systemic diseases with oral manifestations affect the functional ability to eat as well as diet and nutrition status.

Historically, diseases of the oral cavity have been viewed separately from those of the rest of the body. In recent years, however, efforts have been made to recognize oral health as an integral part of overall health.Promotion of oral health has, therefore, been suggested as a way to promote systemic health, since there is a possible role of oral infections as a risk factor for systemic disease. Periodontal diseases are thus postulated as a risk factor for diabetes mellitus and may be related to a number of other systemic diseases, such as chronic obstructive pulmonary disease (COPD), pneumonia, chronic kidney disease, rheumatoid arthritis (RA), cognitive impairment, obesity, metabolic syndrome and cancer. It was shown that treatment of periodontitis in patients with diabetes mellitus leads to an improvement of glycaemic control, and may reduce RA severity and systemic inflammation in Rhuematoid Arthritic patients.

Periodontal diseases in the pathogenesis of preeclampsia

A possible link between periodontitis and preeclampsia was first proposed in 2003, and since then, a considerable amount of studies focused on this association. Although findings were not consistent, there appeared to be an association between periodontitis and preeclampsia. One of the mechanisms involved in this putative relationship may be the systemic nature, i.e. the low grade inflammation in periodontitis. It can be hypothesised that the chronic inflammation of periodontitis superimposes on the already pro-inflammatory state of normal pregnancy. The result may be an abnormally activated inflammatory system that ultimately leads to preeclampsia. Also a direct bacterial infection of maternal organs by invading periodontopathic bacteria,might explain a possible role of periodontitis in the aetiology of preeclampsia.

The involvement of specific periodontal micro-organisms in the pathogenesis of preeclampsia has been examined in some human studies In these studies, a higher prevalence of *P. gingivalis*, *T. forsythia, E. corrodens P. Intermedia* or *A. actinomycetemcomitans* was found in subgingival plaque samples of preeclamptic women as compared with healthy pregnant controls. *P. gingivalis, E. corrodens, T.forsythia, P. intermedia* and *A. actinomycetemcomitans* all produce a variety of proinflammatory factors, including LPS, which may affect the immune response during pregnancy.

Until now, however, there are no studies to substantiate a causal role of periodontal bacteria or bacterial products in the pathogenesis of the maternal syndrome of preeclampsia.

Faas et al established an animal model for preeclampsia, in which an infusion of a low dose of LPS from the enteric bacterium *Escherichia coli* (*E. coli*) into pregnant rats led to activation of the inflammatory response and subsequently to a preeclampsia-like syndrome, characterised by hypertension and proteinuria. This syndrome was pregnancy specific identically treated non-pregnant rats did not develop these symptoms. Therefore it might be possible that the state of pregnancy also influences the susceptibility for periodontopathic micro-organisms,like *P. gingivalis* or its LPS, leading to increased activation of inflammatory cells and pro-inflammatory cytokine production by leukocytes. This may increase the risk on developing endothelial activation/dysfunction and eventually result in the maternal syndrome of preeclampsia.

Periodontal disease is initiated by oral microorganisms, but it is believed that severe periodontal breakdown is mediated by the inflammatory response of the host. The inflammatory response may not be limited to the periodontal area. It has been proposed that daily episodes of bacteremia or dissemination of bacterial endotoxins from the periodontal focus may induce systemic activation of the inflammatory response. Bacteria or bacterial endotoxins in the systemic circulation may induce pro-inflammatory cytokine production. These cytokines then further activate the inflammatory response, which results in a chronic low-grade systemic up-regulation of the inflammatory molecules involving IL-6 and C-reactive protein (CRP) . The inflammatory response also activates inflammatory and endothelial cells and may result in endothelial dysfunction. In pregnancy, the immune response plays a pivotal role in maintaining a healthy equilibrium between the mother and fetal allograft. During a normal pregnancy, the specific immune response is shifted towards a Th2-type immune response, and the inflammatory response is also activated . This activation of the inflammatory response during pregnancy is characterized by the increased expression of activation markers on monocytes and granulocytes, differences in monocyte cytokine production and increased circulating levels of pro-inflammatory cytokines and inflammatory markers, such as CRP.

Studies on association of periodontitis and preeclampsia.

Case control studies

- Castaldi et al examined the periodontal condition of 1562 women within 48 h after delivery. Of the total population included, (10%) women were diagnosed with preeclampsia. The periodontal investigators were blinded for pregnancy outcomes. Gingivitis was found in 34.3% and severe periodontal disease in 17.5% of the women. After adjusting for smoking during pregnancy, no association was found between either gingivitis or severe periodontal disease and preeclampsia. The authors did not report to have adjusted for other confounding factors.

- In a matched case-control study, Canakci et al (Canakci et al. 2004) examined within48 h prior to delivery, the periodontal condition of 41 preeclamptic women and 41normotensive healthy pregnant women. Cases and controls were individually matched for age, parity, gravidity, smoking and prenatal care. Periodontal disease was present in 46.3% of women with preeclampsia and in 21.9% of controls. After adjusting for serum cholesterol levels, serum triglycerides levels and maternal body weight, the conditional multiple logistic regression analysis showed that preeclampsia was associated with periodontal disease.

- Contreras et al53 carried out a case-control study that included 130 pregnant women with preeclampsia and 243 healthy pregnant women. In

both groups the periodontal status was determined between 26 and 36 weeks of pregnancy. Chronic periodontal disease was significantly more prevalent in the preeclamptic group (63.8%) compared with the control group (36.6%). When periodontal disease was further stratified in severity, incipient periodontal destruction was present in 42.3% of cases, compared with 27.2% of controls, whereas moderate-to-severe periodontal destruction was observed in 21.5% of cases, compared with 9.5% of controls. The authors did not report to have adjusted for confounding factors.

- Khader et al conducted a blinded case-control study amongst 115 preeclamptic women and 230 healthy controls within 24 h after delivery. Only those, non-smoking and non-alcohol drinking women were included in this study. After confounding for maternal age, parity, (family) history of preeclampsia, family history of cardiovascular disease, pre-pregnancy BMI, twin birth and self-reported emotional stress during pregnancy, no statistical differences were found between cases and controls concerning any of the eight periodontal parameters investigated.

- The association between periodontal disease and early-onset preeclampsia (<34 weeks of pregnancy) was tested in a case-control

study The periodontal condition of 17 early-onset preeclamptic women and 35 women with uncomplicated pregnancies was examined in a period of 3-28 months postpartum. Severe periodontal disease was present in 82% of the post-preeclamptic women and in 37% of the controls. After adjusting for age, BMI, smoking and educational level, severe periodontal disease was associated with preeclampsia.

- The aim of a case-control study performed by Canakci et al was to correlate the severity of periodontal disease to the severity of preeclampsia. Dental and periodontal examinations were performed in 20 mild preeclamptic, 18 severe preeclamptic and 21 healthy pregnant women within 48 h preceding delivery. Mild periodontal disease was found in 16.7% of the severe preeclamptic women, in 25% of the mild preeclamptic women and in 28.6% of the controls. Severe periodontal disease was detected in 72.2% of the severe preeclamptic women, in 50% of the mild preeclamptic women and in 33.3% of the controls. After adjusting for age, smoking, body weight, socioeconomic status and educational level, the results showed that severe periodontal disease wasassociated with both mild and severe preeclampsia.

- Siqueira et al performed a matched case-control study on 125 preeclamptic women and 375 healthy controls within 48 h after delivery. The frequency of periodontal disease before matching was significantly

higher among the preeclamptic women (56.7%) than among the control group (39%). After matching and adjusting for maternal age ≥30 years, chronic hypertension, primiparity, previous pre-term birth and prenatal visits, periodontal disease remained associated with preeclampsia. However, when periodontal probing depth (PPD) and clinical attachment level (CAL) were tested with cut-off points of ≥5 mm or ≥7 mm, the ORs for preeclampsia were not significant, indicating that periodontal breakdown in itself was not associated with preeclampsia in this study.

- A recent epidemiological study was conducted by Lohsoonthorn et al, whoexamined the periodontal condition of 150 preeclamptic and 150 healthy controls within 48 h after delivery. In the preeclamptic group, 49.3% of the women had mild periodontitis, 21.3% had moderate periodontitis and severe periodontitis was present in 8.0% of the women. In the control group, 54.0% of the women were diagnosed with mild periodontitis, while moderate periodontitis was present in 20.7% and severe periodontitis in 4.7% of the women. After adjusting for maternal age, educational attainment, parity, pre-pregnancy BMI, annual household income, employment during pregnancy, marital status, onset of pre-natal care, alcohol use and smoking during pregnancy, no association between periodontal disease and preeclampsia was found.

- In a study conducted by Nabet et al, the association between periodontitis and pre-term delivery (<37 weeks of gestation) was analysed according to the causes of preterm birth. For this purpose, 1108 cases with pre-term delivery (liveborn child between 22-36 weeks of gestation) and 1094 controls with deliveries at term (≥37 weeks of gestation) were included in the study. One hundred and ninety-eight (18.1%) of the cases were induced pre-term deliveries due to preeclampsia. Periodontal examinations were performed within 2-4 days after delivery. Localized periodontitis was found in 13.6% of the cases and in 10.8% of the controls, while generalized periodontitis was present in 20.7% of the cases and in 10.8% of the controls. After adjusting for maternal age, parity, nationality, educational level, marital status, employment during pregnancy, BMI before pregnancy and smoking status, an association was observed between generalized periodontitis and induced pre-term birth due to preeclampsia.

- The most recent case-control study conducted by Shetty et al, examined the periodontal condition of 30 preeclamptic women and 100 healthy pregnant controls at recruitment (26-32 weeks of gestation) and within 48 h after delivery. At enrolment, 100% of the cases and 78% of the controls were diagnosed with periodontal disease (CAL ≥3 mm). After adjusting for maternal age, body weight, occupation, education and

income, severe periodontal disease (CAL >5 mm) both at enrolment as well as at delivery was associated with an increased risk of preeclampsia. There were no significant differences in disease progression between cases and controls.

Cohort studies

As part of a prospective cohort study on the effect of maternal periodontal disease on obstetric outcomes (the Oral Conditions and Pregnancy Study, OCAP, Boggess et al examined the periodontal condition of 850 pregnant women at enrolment (before 26 weeks' gestation) and followed them until delivery. Thirty-nine women (4.6%) developed preeclampsia. At enrolment, 58.4% of the women were diagnosed with mild periodontitis and 14.7% had severe disease. In order to determine periodontal disease progression, a second periodontal examination was performed in 763 of the women within 48 h after delivery. At this time, 37.3% of the women had mild periodontal disease and 13.1% had severe periodontal disease. Periodontal disease progression occurred in 26.6% of the women. After adjusting for maternal age, race, delivery at <37 weeks' gestation and smoking during pregnancy, the authors found that women were at a higher risk of developing preeclampsia if they had severe periodontal disease at delivery or periodontal disease progression during pregnancy. Periodontal disease at enrolment, however, was not associated with an increased risk of developing preeclampsia.

As part of a large multi-centre prospective cohort study (the Periodontal Infection and Prematurity Study, PIPS), the risk of adverse pregnancy outcomes in women with periodontal disease compared with those without disease was assessed by Srinivas et al. In this study, 311 pregnant women with periodontal

disease between 6 and 20 weeks of gestation and 475 without periodontal disease were included. Periodontal examinations were performed by trained nurses. Sixteen women (5.2%) with periodontal disease developed preeclampsia, while in the periodontally healthy group 32 women (6.7%) developed preeclampsia. After adjusting for maternal age, race, tobacco, obesity and chronic hypertension, the authors found no association between the presence of periodontal disease during pregnancy and preeclampsia.

RCTs

In an intervention study conducted by Michalowicz et al, the effect of periodontal treatment on pregnancy outcomes was examined. Eight hundred and twelve pregnant women with periodontal disease were randomly assigned to two groups between 13 and 17 weeks of gestation. Periodontal disease was assessed at trial entry, at 21-24 weeks' gestation and at 29-32 weeks' gestation. The treatment group received periodontal therapy before 21 weeks of gestation, which consisted of up to four sessions of scaling and rootplaning (SRP). Treatment participants also received monthly tooth polishing, oral hygiene instruction and if needed, SRP was provided to the treatment group until delivery. The control group received only a brief examination at monthly follow-ups and received periodontal treatment after delivery. Although the primary outcome of this study was gestational age at the end of pregnancy (pre-term birth), the authors also evaluated preeclampsia as one of the secondary outcomes. Periodontal treatment during pregnancy did not significantly alter the rate of preeclampsia, despite the improvement of the periodontal status.

MATERIALS AND METHODS

MATERIALS AND METHODS

The present study was conducted at Institute of at Madras Medical College, Chennai Tamilnadu during the academic year 2017. The study was done in 200 patients for duration of 8 months. Participants were informed about the aims of the study and a written informed consent was obtained from them.

An eligible sample was selected based on the following criteria.

Inclusion criteria

Women of age 18–35 years who gave birth to live infants in the hospital unit.

Exclusion criteria

- Women who gave birth to more than one infant in a single delivery;
- those who had undergone *in vitro* fertilization;
- those who were suffering from any systemic diseases before pregnancy,
- those with history of placental, cervical, and/or uterine abnormalities;
- those who had human immunodeficiency virus infection;
- those who were on antibiotic prophylaxis for dental treatment;

Study design

During the study period, 200 women who were admitted in the obstetric department were analysed. Periodontal examination was performed 48 h after delivery, but before assessment of medical records to avoid the occurrence of

bias. Intraoral examination was done with the help of artificial light source, mouth mirror, William's periodontal probe, and cotton pliers. The following clinical parameters were used to evaluate the clinical signs of inflammation and periodontal tissue destruction:

Gingival bleeding index

The gingival bleeding was determined dichotomously by gentle probing of the gingival crevice with the William's periodontal probe. The appearance of bleeding within 10 s indicated a positive score, which was expressed in percentage.

Probing pocket depth

The distance between the base of the pocket and the gingival margin was recorded for each individual tooth.

Clinical attachment level

This is determined by measuring the distance from the cemento-enamel junction to the probable base of the sulcus. Teeth were excluded if their cemento-enamel junction was not identifiable, with large undefined restorations, large carious lesions or fractures, or if they were erupting.

For the purpose of analysis, maternal periodontitis was defined as probing depth ≥4 mm and clinical attachment level (CAL) loss ≥3 mm at the same site in at least four teeth.

Women were diagnosed with preeclampsia if they had the following:

- Blood pressure ≥140/90 mm Hg on two separate occasions after week 20 of gestation.
- Proteinuria defined as protein concentration ≥0.30g/dl in 24 hours urine collection.

Medical data

Demographic data, medical history, and detailed information on events during pregnancy and delivery were obtained from patients' medical records. Medical data were reviewed by an obstetrician to confirm the inclusion and exclusion criteria.

After assessment of the medical records, the women were first divided as follows:

Group A: Case group consisting of 100 preeclamptic women

Group B: Control group consisting of 100 non-preeclamptic women.

Statistical analysis was carried out to check the significance of maternal periodontitis in these two groups.

OBSERVATION AND ANALYSIS

OBSERVATION

The study undertaken here is an observational case control study conducted at obstetrics and gynaecology department of Madras Medical College.

Total number of patients analysed : 200

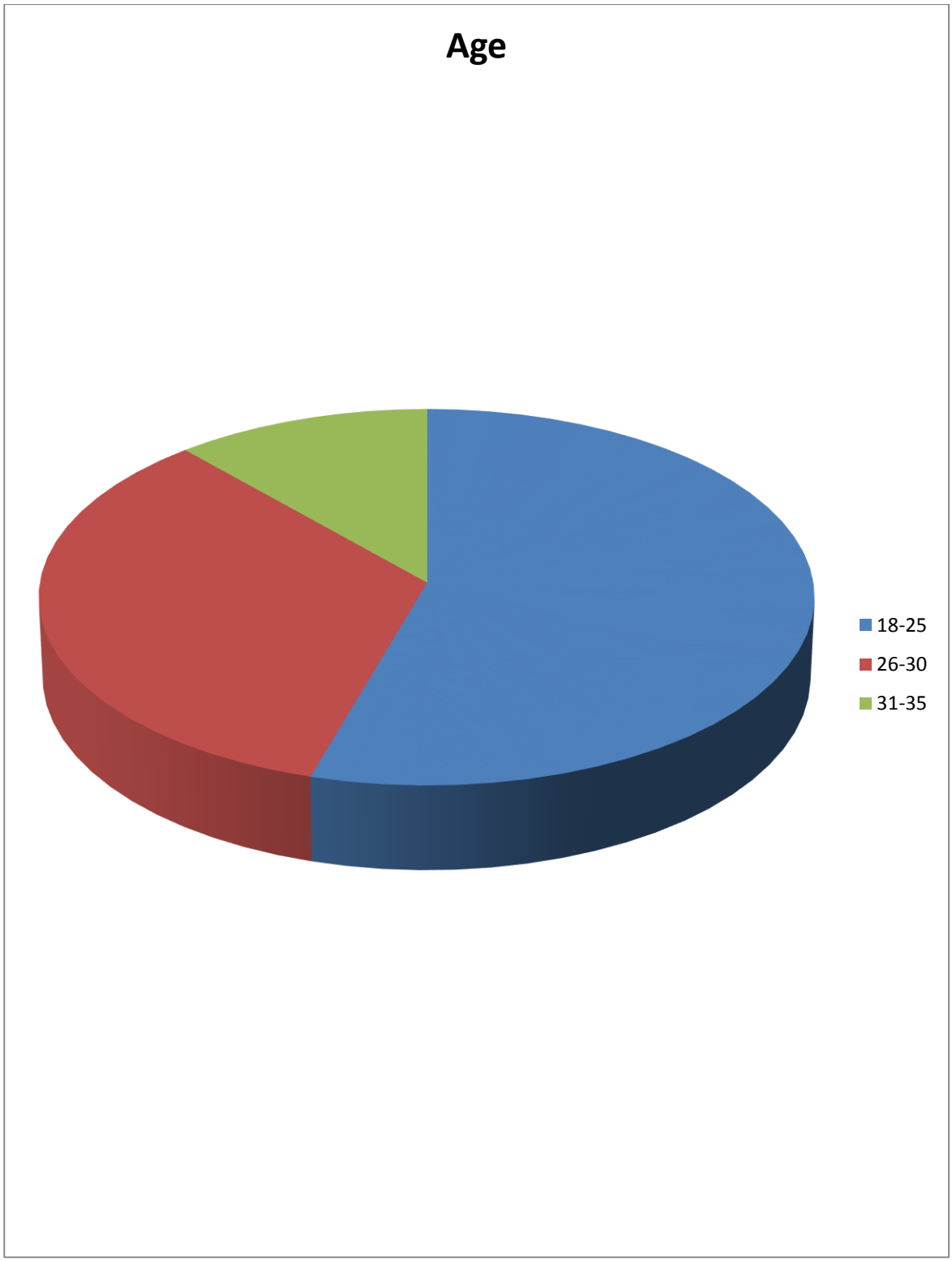
The patients were divided into 2 groups, cases and controls. 100 patients who had preeclampsia and 100 non preeclamptic patients. These patients were subjected to dental examination 48 hrs after delivery and were diagnosed to have periodontitis or not.

OBSERVATION AND ANALYSIS

AGE DISTRIBUTION

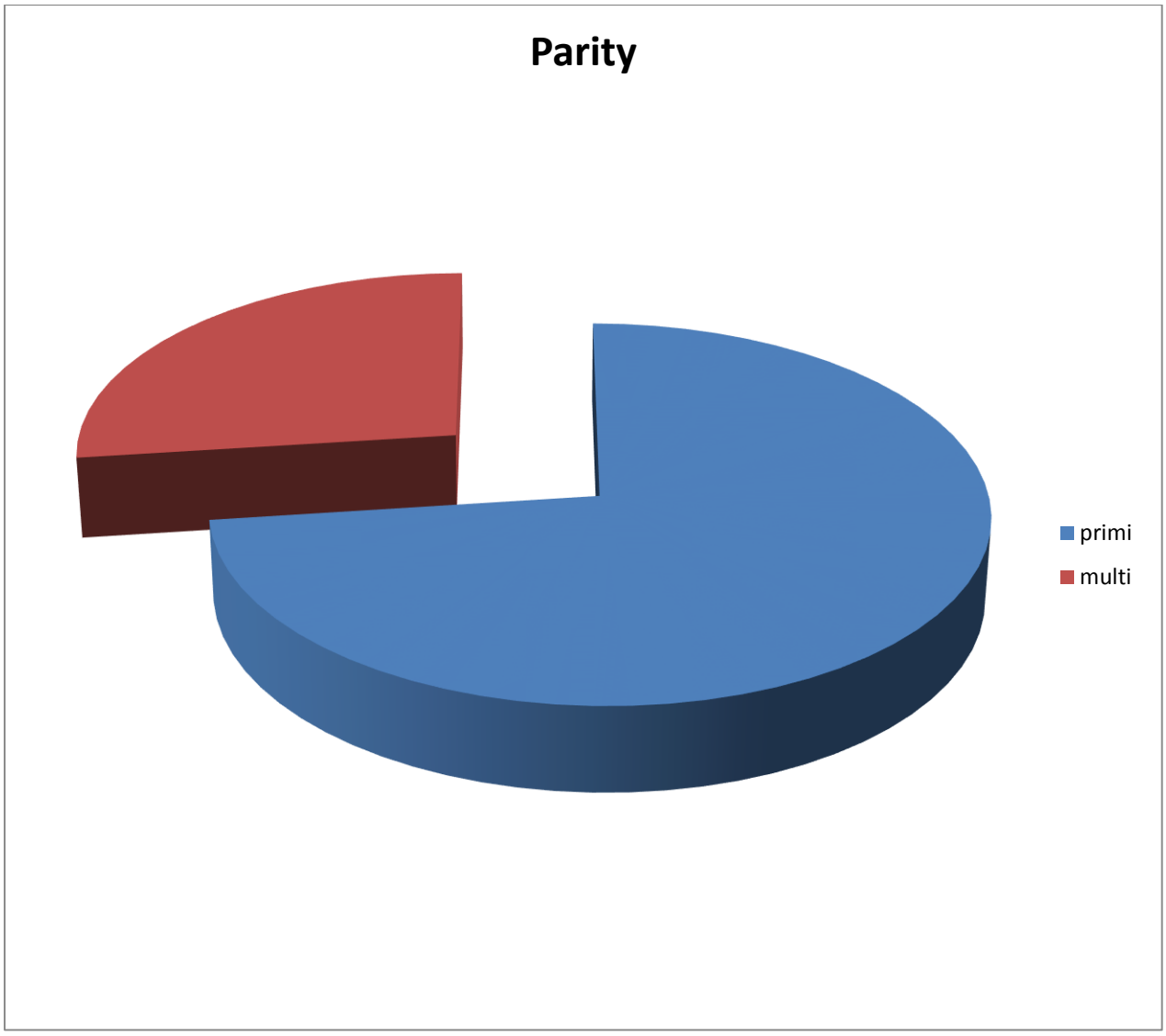
AGE GROUP	NO.OF PATIENTS	PERCENTAGE
18-25	109	54.5
26-30	68	34
31-35	23	11.5

Among 200 patients included in the study 109 were between the age 18-25 yrs,68 were between 26-30yrs and 23 were between 31-35yrs.


Age
18-25
26-30
31-35

PARITY:

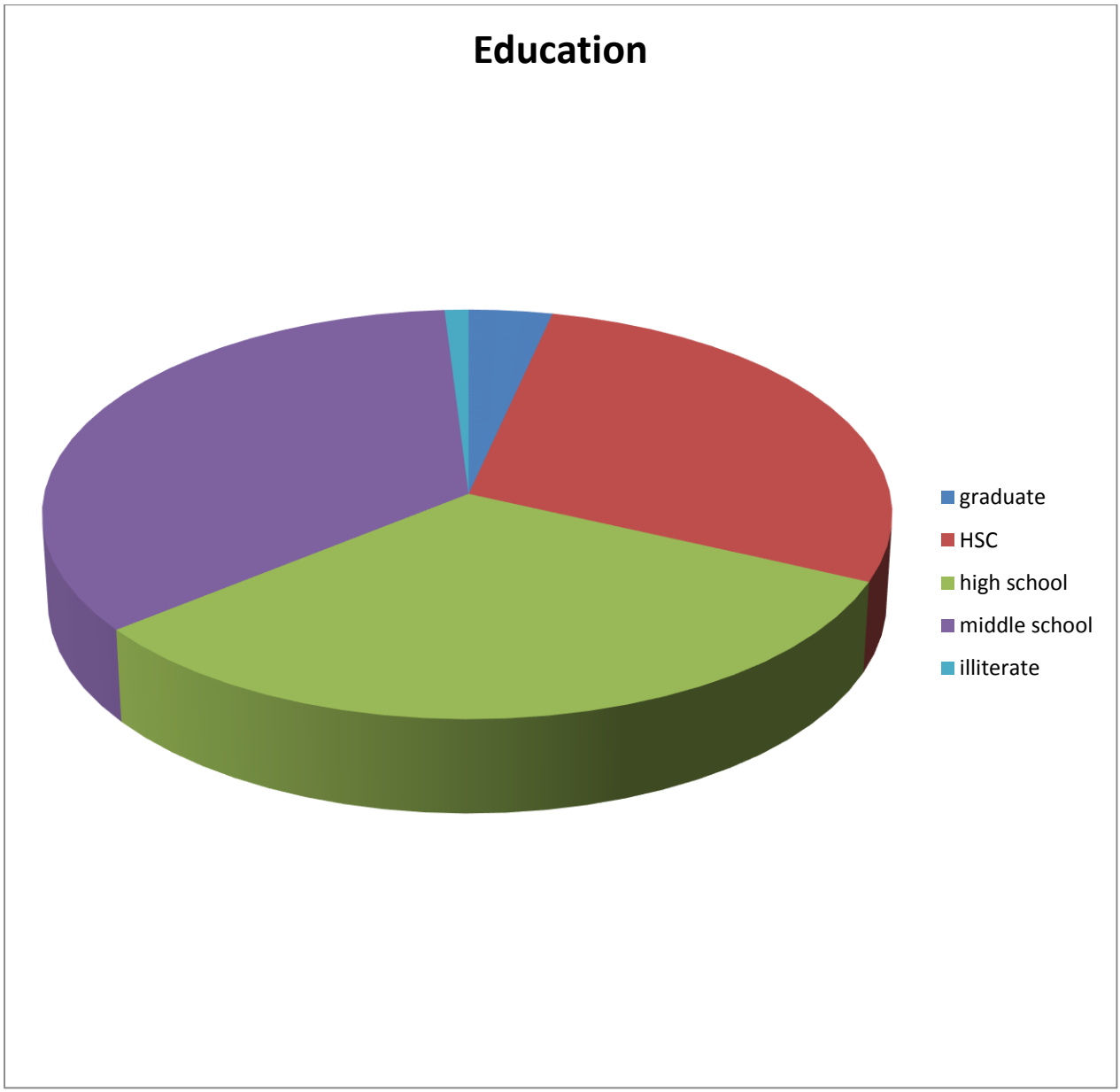
PARITY	NO.OF PATIENTS	PERCENTAGE
PRIMIGRAVIDA	146	73
MULTIGRAVIDA	54	27
TOTAL	200	100



In the study of 200 patients 146 patients were P1L1 and 54 were Multipara.

EDUCATIONAL STATUS

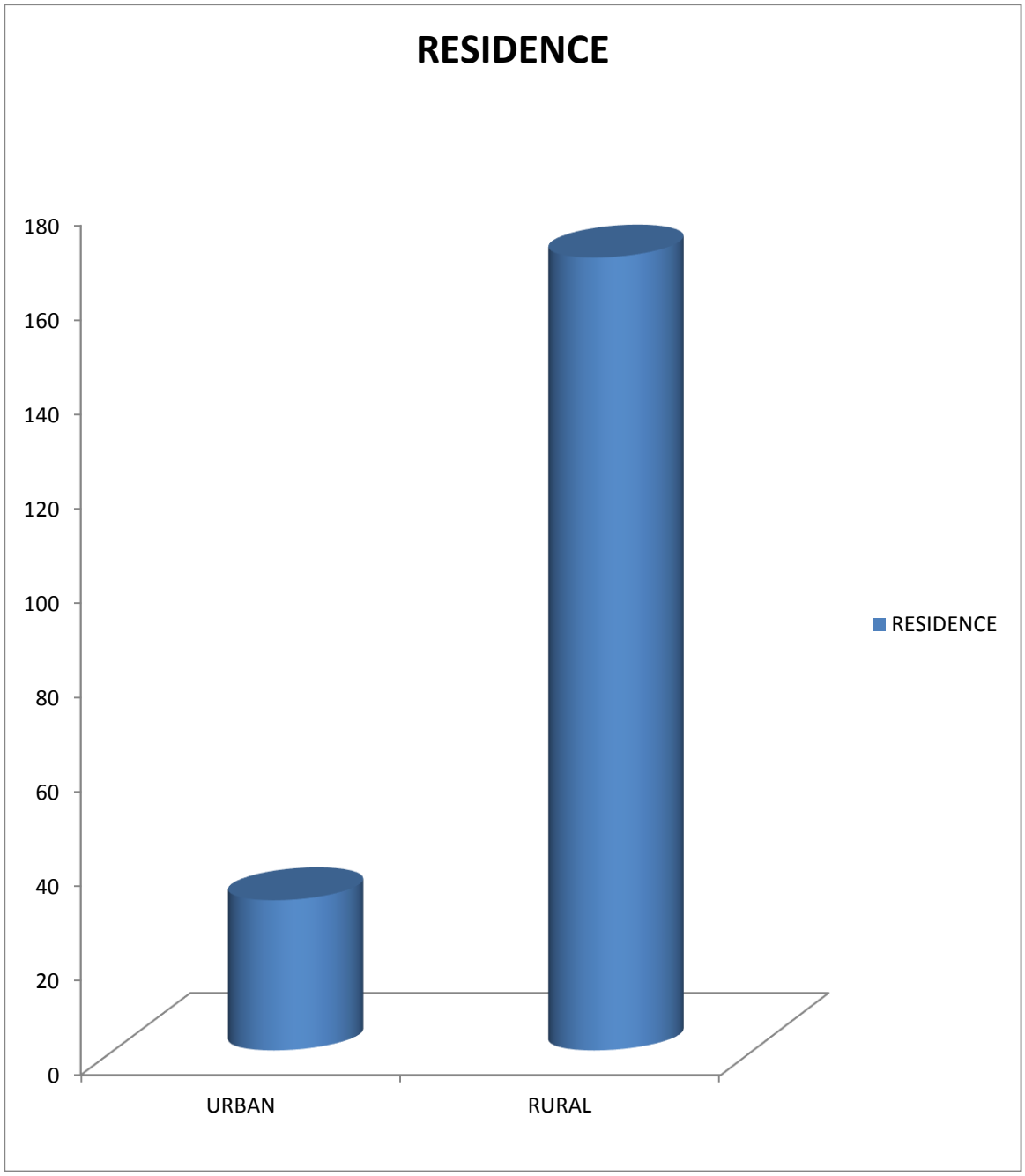
EDUCATION	NO.OF PATIENTS	PERCENTAGE
GRADUATE	7	3.5
HIGHER SECONDARY	57	28.5
HIGH SCHOOL	65	32.5
MIDDLE SCHOOL	70	35
ILLITERATE	2	1



Nearly 35% of study population has stopped education at the middle school level. So awareness about dental hygiene among pregnant ladies should be brought in.

RESIDENCE

RESIDENCE	NO.OF PATIENTS	PERCENTAGE
URBAN	31	15.5
RURAL	169	84.5
TOTAL	200	100

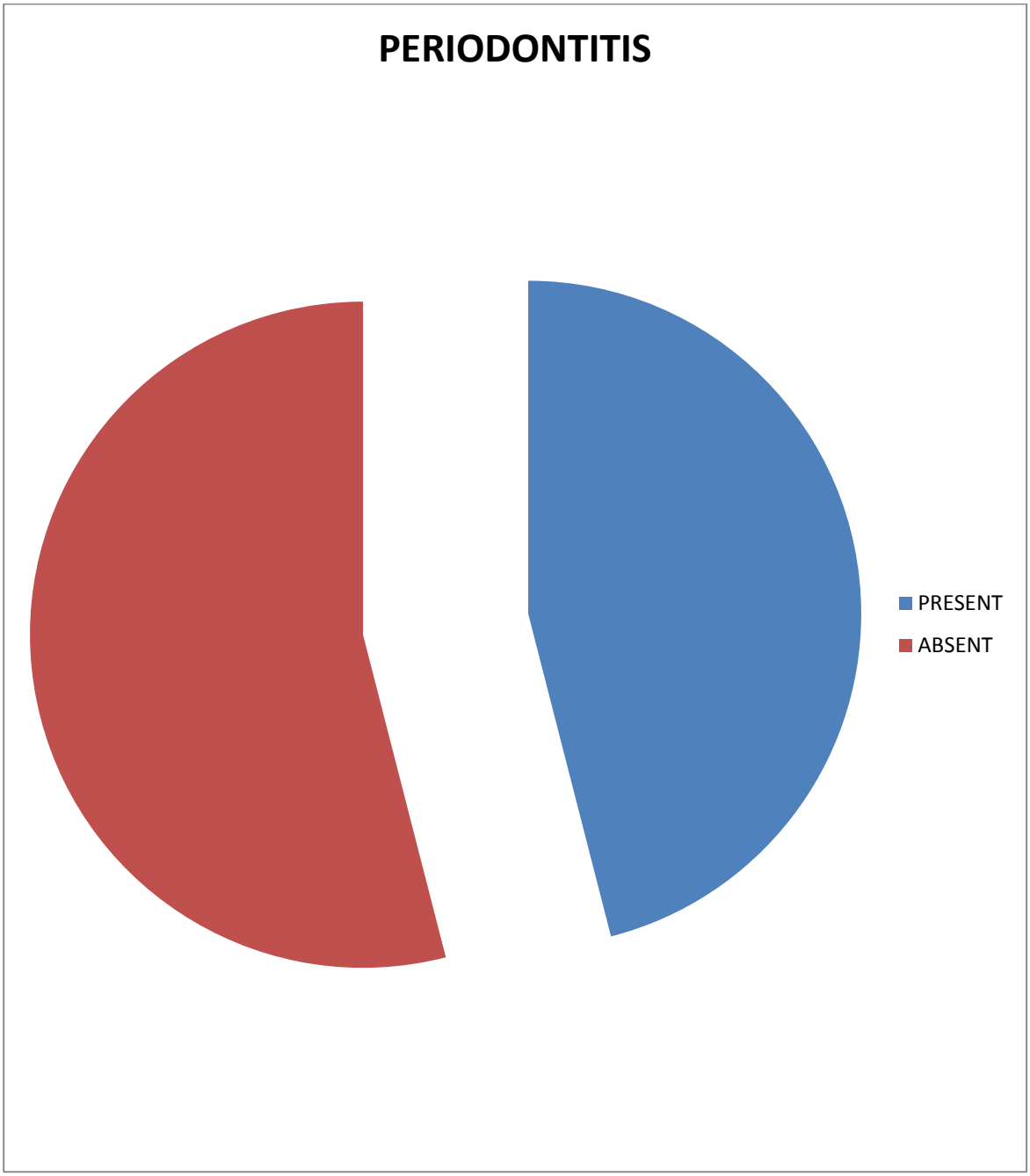


Majority i.e 84% of study population belongs to rural area. Oral hygiene should be emphasised among these people

PATIENTS WITH PERIODONTITIS

PERIODONTITIS	NO.OF PATIENTS	CASES	CONTROL	PERCENTAGE OF PTS WITH PERIODONTITIS
PRESENT	92	67	25	46
ABSENT	108	33	75	54
TOTAL	200	100	100	100

Out of 200 patients analysed 92 patients were diagnosed to have periodontitis and severe.which included mild ,moderateand 108 patients were disease free. Out of 92 patients 67 patients had preeclampsia and periodontitis


PERIODONTITIS
PRESENT
ABSENT

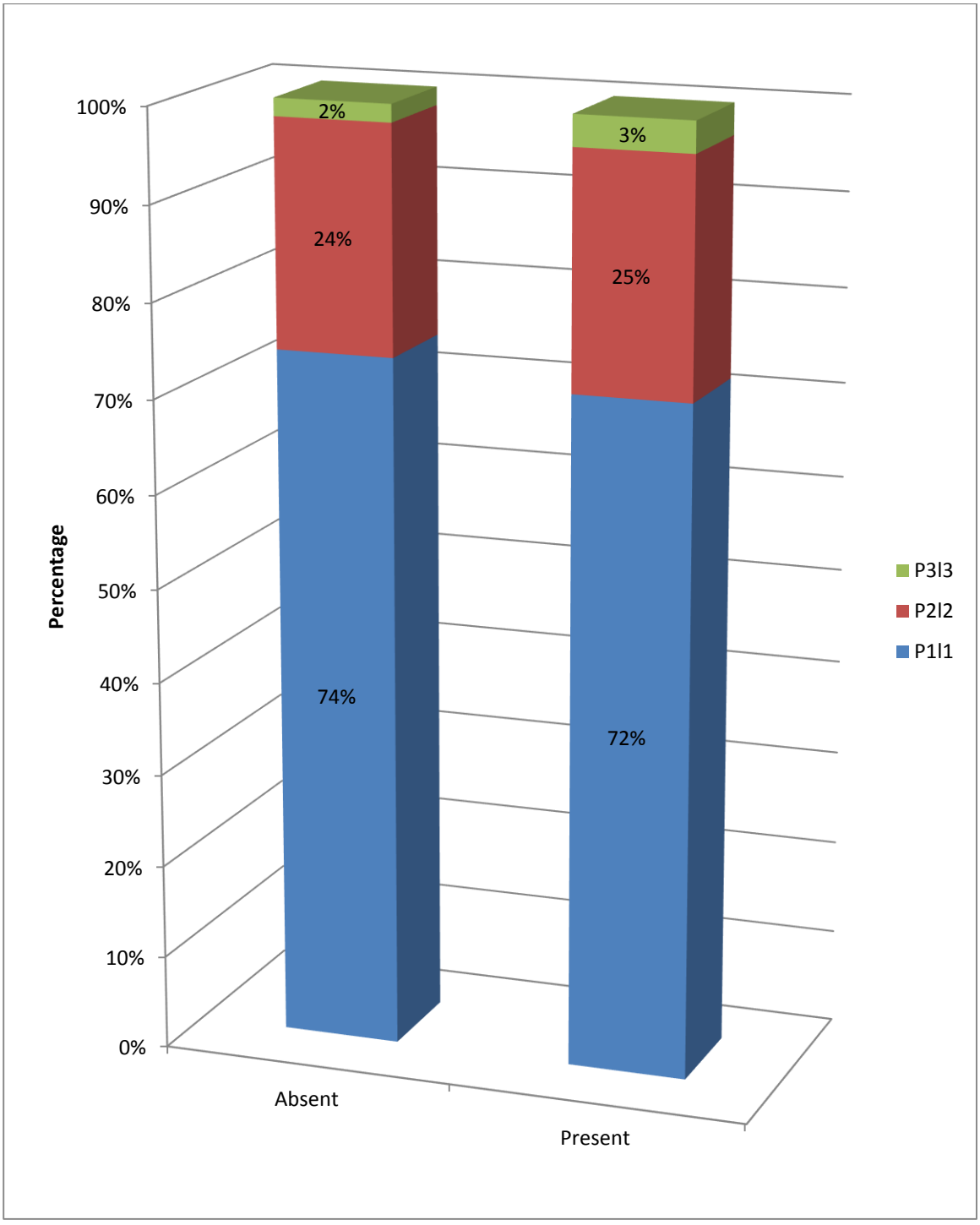
Association of periodontitis and parity

Crosstab

			periodontis_score		Total
			Absent	Present	
Parity	P1l1	Count	80	66	146
		% within periodontis_score	74.1%	71.7%	73.0%
	P2l2	Count	26	23	49
		% within periodontis_score	24.1%	25.0%	24.5%
	P3l3	Count	2	3	5
		% within periodontis_score	1.9%	3.3%	2.5%
Total		Count	108	92	200
		% within periodontis_score	100.0 %	100.0%	100.0%

Pearson Chi-Square=0..449 p=0.799 (no significance)

The analysis shows no significant difference in association between parity and periodontitis

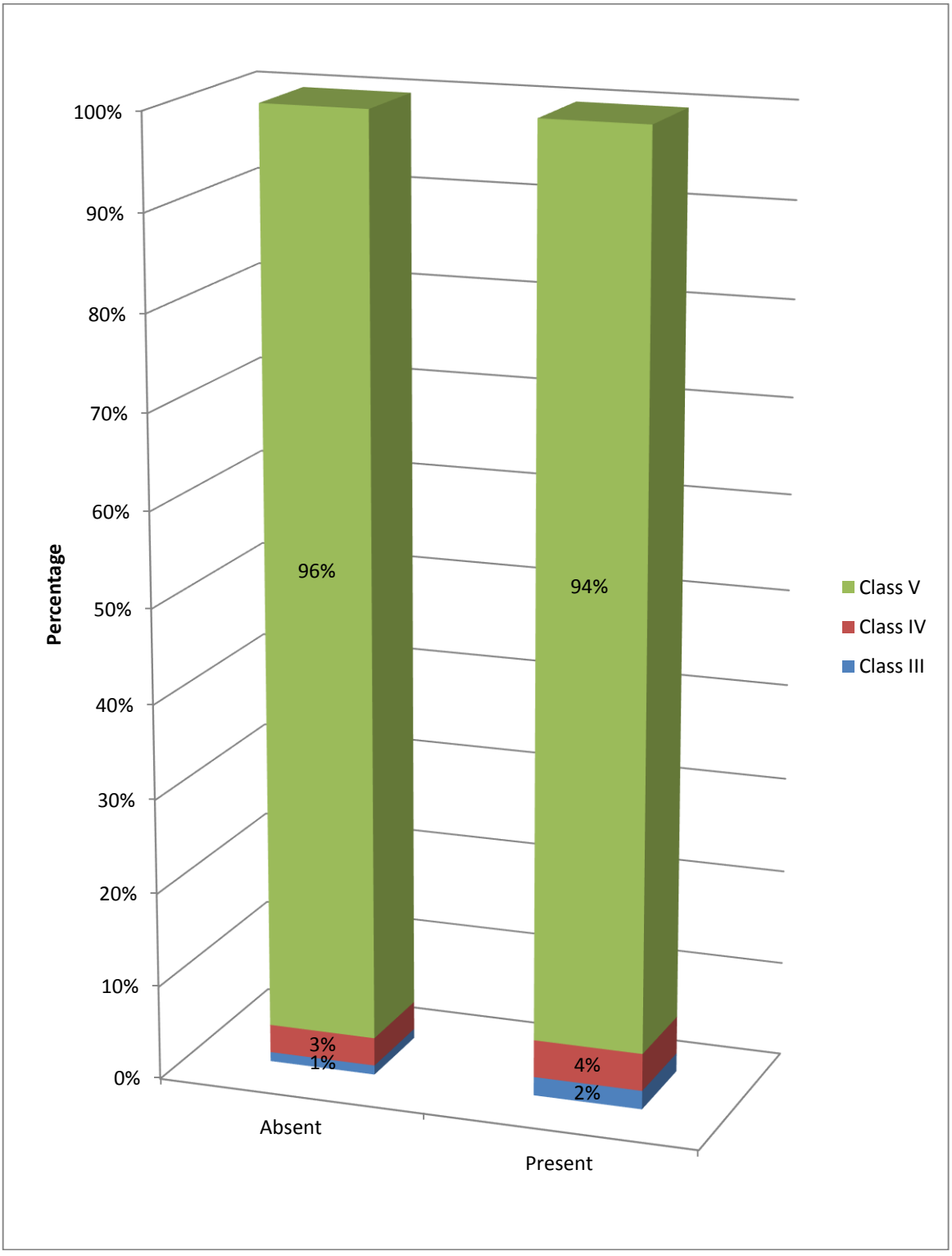

100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
Percentage
2%
24%
74%
3%
25%
72%
Absent
Present
P3I3
P2I2
P1I1

Association of periodontitis and socioeconomic class

Crosstab

			periodontis_score		Total
			Absent	Present	
Socioeconomic_class	Class III	Count	1	2	3
		% within periodontis_score	0.9%	2.2%	1.5%
	Class IV	Count	3	4	7
		% within periodontis_score	2.8%	4.3%	3.5%
	Class V	Count	104	86	190
		% within periodontis_score	96.3%	93.5%	95.0%
Total		Count	108	92	200
		% within periodontis_score	100.0 %	100.0%	100.0%

Pearson Chi-Square=0..907 p=0.635


100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
Percentage
96%
94%
3%
1%
4%
2%
Absent
Present
Class V
Class IV
Class III

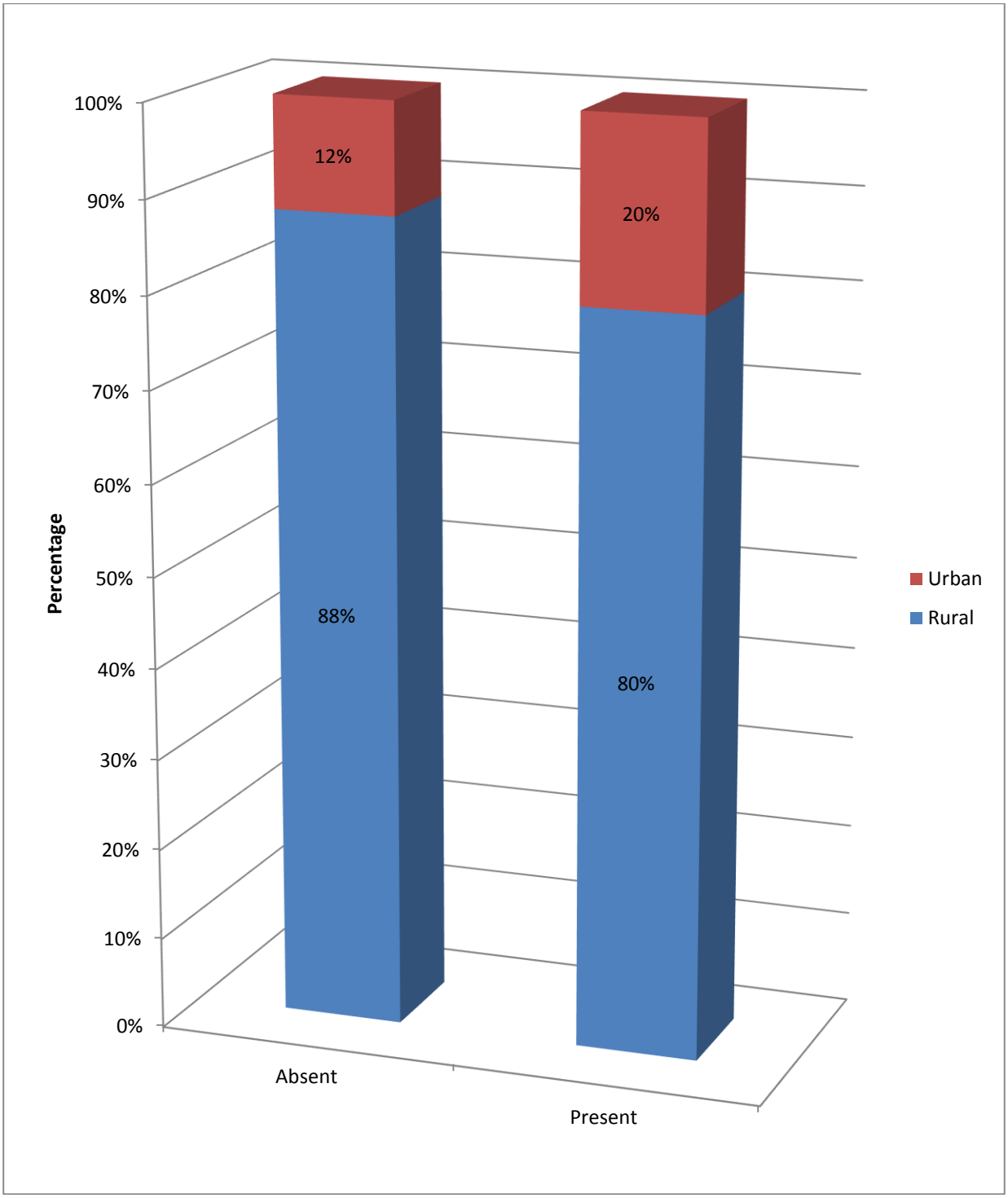
Association of periodontitis and residence

Crosstab

			periodontis_score		Total
			Absent	Present	
Residence	Rural	Count	95	74	169
		% within periodontis_score	88.0%	80.4%	84.5%
	Urban	Count	13	18	31
		% within periodontis_score	12.0%	19.6%	15.5%
Total		Count	108	92	200
		% within periodontis_score	100.0 %	100.0%	100.0%

Pearson Chi-Square=2.150 p=0.143

Majority of study population belong to rural area. The study shows no significant association between periodontitis and residence


100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
Percentage
12%
88%
20%
80%
Absent
Present
Urban
Rural

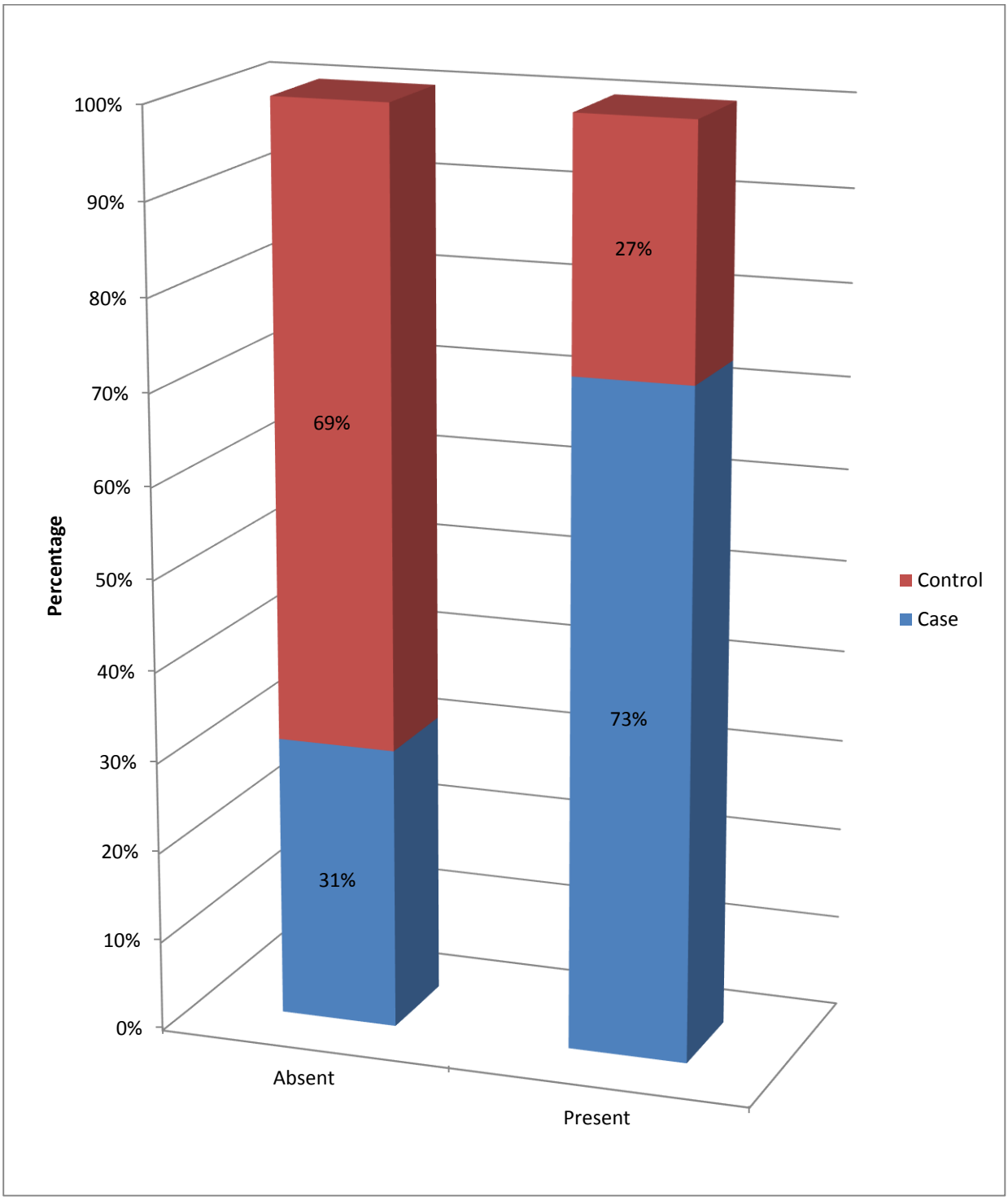
Association of periodontitis and preeclampsia

periodontis_score Crosstabulation

			periodontis_score		Total
			Absent	Present	
group –	Case	Count	33	67	100
		% within periodontis_score	30.6%	72.8%	50.0%
	Control	Count	75	25	100
		% within periodontis_score	69.4%	27.2%	50.0%
Total		Count	108	92	200
		% within periodontis_score	100.0%	100.0%	100.0%

Pearson Chi-Square=35.507** p<0.001(significant)

The analysis shows significant association between periodontitis and preeclampsia


100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
Percentage
69%
31%
27%
73%
Absent
Present
Control
Case

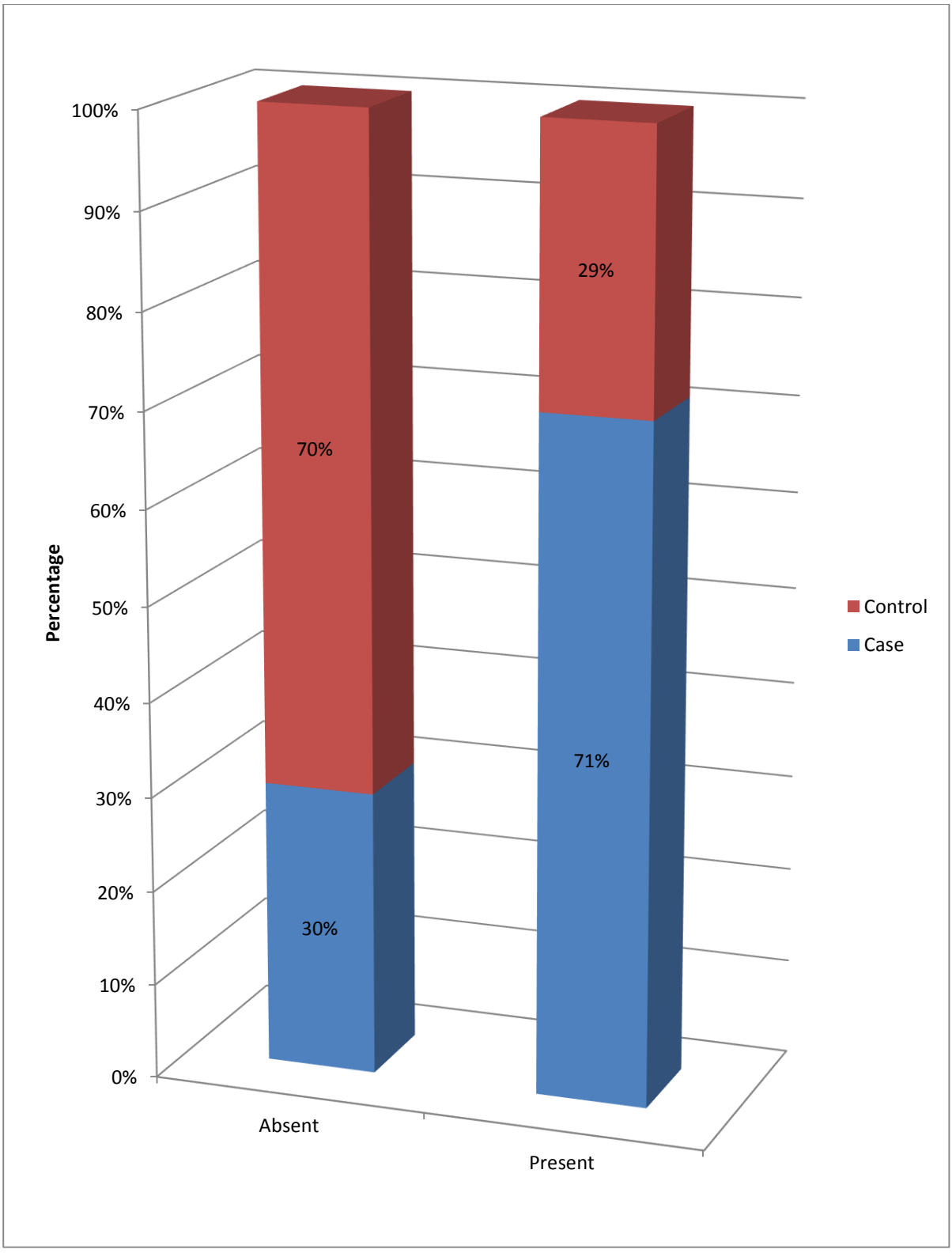
Association of periodontitis and preeclampsia after matching for primi parity.

out of 146 primi parity association for case and control for periodontitis

group_ * periodontis_score Crosstabulation

			periodontis_score		Total
			Absent	Present	
group_	Case	Count	24	47	71
		% within periodontis_score	30.0%	71.2%	48.6%
	Control	Count	56	19	75
		% within periodontis_score	70.0%	28.8%	51.4%
Total		Count	80	66	146
		% within periodontis_score	100.0 %	100.0%	100.0%

Pearson Chi-Square=24.588** p<0.001 (significant)



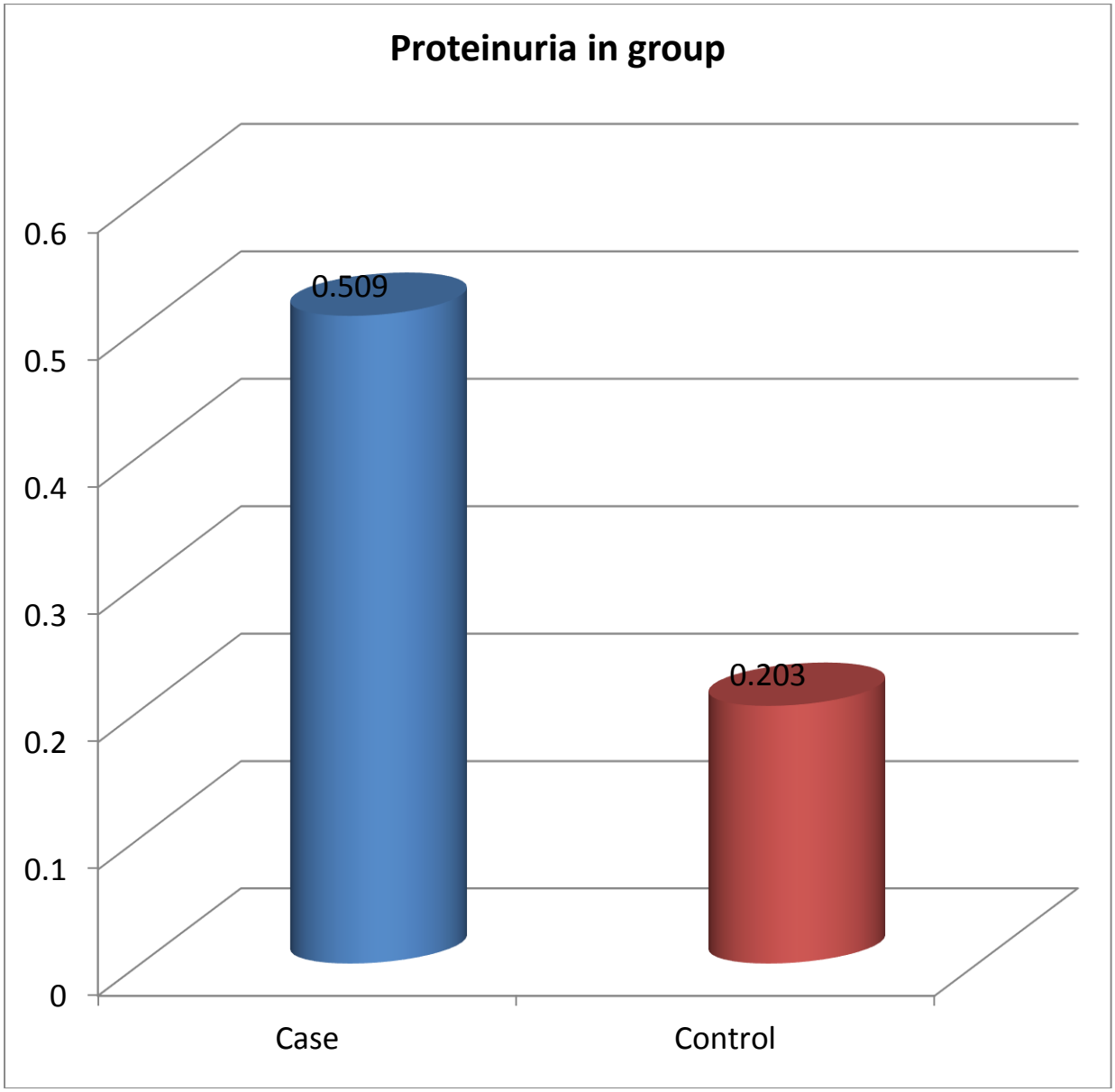
After matching for primi parity there was significant association between periodontitis and preeclampsia. Matching for primi parity was done since it was a significant confounding factor

Comparison of proteinuria in group

Group Statistics

	group_	N	Mean	Std. Deviation	Std. Error Mean	
Proteinuria	Case	100	.5090	.13566	.01357	20.028**
	Control	100	.2030	.07029	.00703	

**p<0.001(significant)

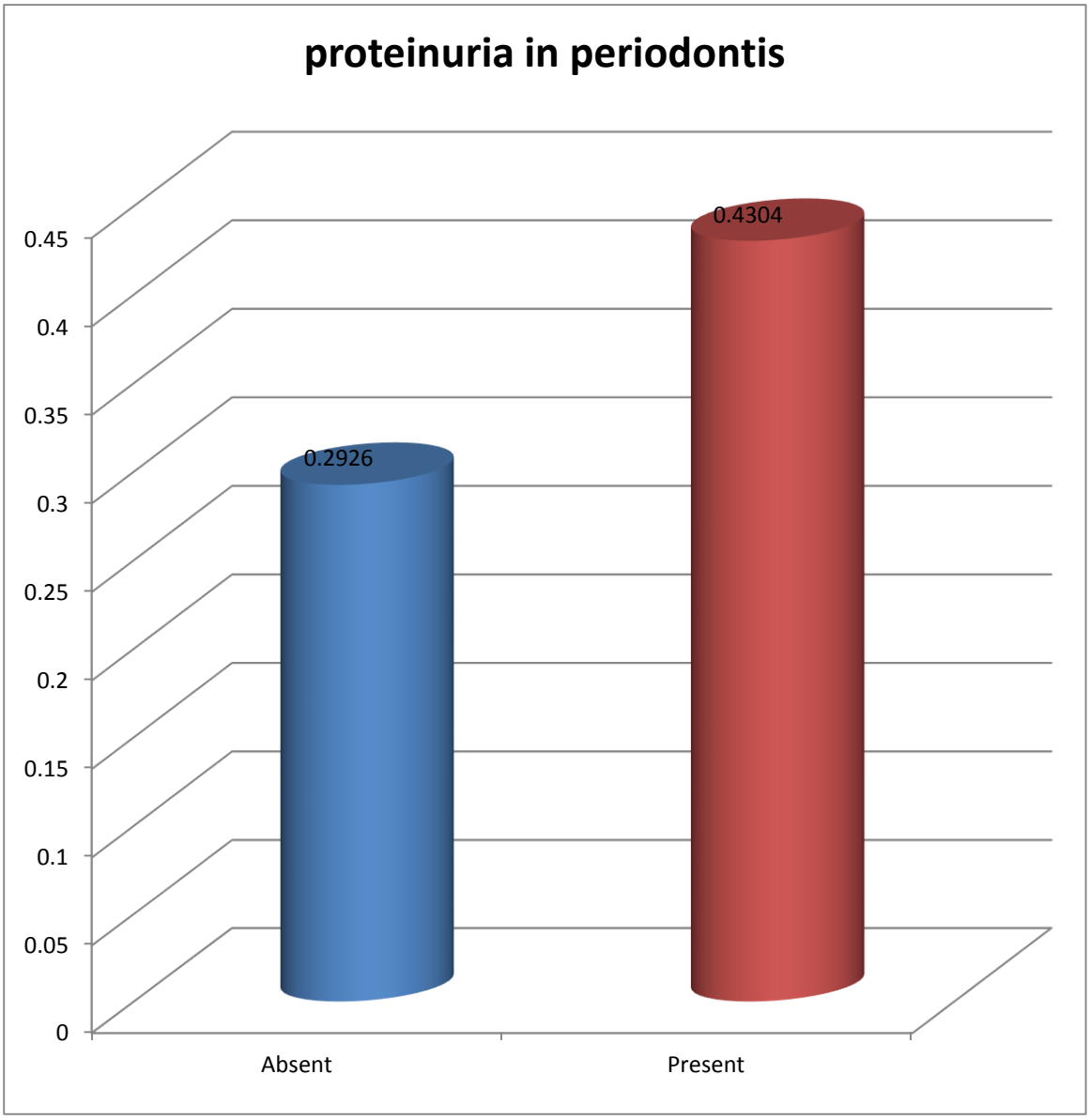

Proteinuria in group
0.6
0.5
0.4
0.3
0.2
0.1
0
0.509
0.203
Case
Control

comparison of proteinuria in periodontis

Group statistics

	Periodontis score	N	Mean	Std. Deviation	Std. Error Mean	
Proteinuria	Absent	108	.2926	.15926	.01533	5.559**
	Present	92	.4304	.19143	.01996	

**p<0.001 significant


proteinuria in periodontis
0.45
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0.2926
0.4304
Absent
Present

DISCUSSION

Discussion

Periodontal disease is a disease characterized by intermittent periods of activity and inactivity that initiates local inflammation leading to tissue destruction. Many authors like Offenbacher *et al*, Lin *et al*, and Boggess *et al*.have emphasized the role of active periodontal disease causing translocation of periopathogenic bacteria to the fetal-placental unit, resulting in placental damage and manifestations of preeclampsia.

The mechanisms leading to preeclampsia have been speculated to be because of impaired placental perfusion leading to an insufficient placental implantation. Endothelial cell dysfunction caused by cytokines like TNF-α and oxidative stress lead to changes in the structure of placental bed as well as uterine blood vessels, resulting in high levels of fibronectin, endothelin, tissue plasminogen activator, Von Willebrand factor, and thrombomodulin in the maternal blood. Levels of free 8-isoprostane, which is a marker of lipid peroxidation and a strong vasoconstrictor, are found to be increased in preeclamptic women. The burden of endotoxins, inflammatory cytokines, and oxidative stressors at the level of maternal–fetal unit can also be increased by periodontitis. Potent vascular stressors like soluble intercellular adhesion molecules (sICAMs) are found to be increased in periodontitis, thus compounding preeclampsia. So, any maternal infection or inflammatory disease like periodontitis and atheroma formation can initiate and propagate acute uteroplacental atherosis and preeclampsia.

Oral cavity and systemic diseases form a two-way street, i.e. both entities can

affect each other. Golub *et al*. proposed a “two-hit” model for chronic periodontitis and systemic diseases like arthritis and osteoporosis. They concluded that the periodontopathic bacteria provided one “hit,” whereas systemic inflammations elevating the levels of pro-inflammatory biomarkers like C-reactive protein (CRP), IL-6, and matrix metallopeptidase-9 (MMP-9) in serum or plasma act as a second “hit.” It is possible that preeclampsia could also lead to an aggravation of pre-existing periodontal problems or even co-induce periodontal destruction, but further studies regarding this co-relation have to be carried out.

The aim of the present study was to assess the association between maternal periodontitis and preeclampsia.

In this observational study involving 200 patients ,100 patients were with preeclampsia and 100 were without preeclampsia. All the 200 patients were subjected to dental examination 48hrs post delivery and 92 patients were found to have periodontitis.

146 patients out of 200 patients were P1L1 and majority of study population belonged to the age group of 18-25 years. Most of the study population belonged to rural area and from class V socioeconomic status.

Of the 92 patients with periodontitis 67 patients were with preeclampsia and 25 patients without preeclampsia. Statistical analysis were carried out and this showed a significant association between periodontitis and preeclampsia.

This study also showed significant association between periodontitis and preeclampsia after matching for primiparity.

Comparison of preeclamptic group and normotensive group showed no significant risk association between maternal age, parity ,area of residence and socioeconomic status.

SUMMARY

In the present study 200 patients were analysed for periodontitis out of which 100 were preeclamptic and other 100 kept as control were normotensive.

Age group of the study population was between 18-35years and majority of the patients were between the age of 18-25years. Study popultation were mostly from rural area and from class V socioeconomic status.

35% of study population had stopped schooling from middle standard.

Most of the study population were P1L1.

Of the 200 patients analysed 92 were found to have periodontitis, which accounts for 46% of study population. Out of the 92 patients having periodontitis 67 patients were preeclamptic.

The study showed no significant association between age, parity,socioeconomic status and place of residence to periodontitis.

There is significant association between maternal periodontitis and preeclampsia.

After matching for primiparity also there was significant association etween periodontitis and preeclampsia.

This study shows a positive association between periodontitis and preeclampsia. Hence periodontits may be considered as a risk factor for preeclampsia.

CONCLUSION

The present study showed significant association between periodontitis and preeclampsia.

In the study there was significant association between primi parity and Preeclampsia. But after matching for primiparity, maternal periodontitis still remained a significant factor in the preeclamptic group.

Comparison of preeclamptic group and normotensive group showed no significant risk association between maternal age,parity ,socioeconomic status and area of residence.

In the present study, maternal periodontitis is associated with an elevated risk for preeclampsia. This finding is consistent with the findings of Boggess *et al*., Canakci *et al* .,Contreras *et al*., Cota *et al*., and Siqueira *et al*., and is contrary to the study findings of Khader *et al*. and Rai, who found no relation between maternal periodontitis and preeclampsia. The slight variation in the present study could be related to biases introduced by sample size and heterogeneity of the criteria to define periodontitis.

The association between periodontitis and preeclampsia has been supported by the hypothesis that chronic periodontitis infection increases the risk of developing preeclampsia in pregnant women. The pathogenesis of periodontitis and preeclampsia is multifactorial and similar in many ways. So, periodontal treatment should be considered for reducing the risk of preeclampsia.

At the first prenatal visit, health care providers should assess a woman's oral health. A simple approach to prenatal assessment can be accomplished by using the questions provided here

1) Do you have swollen or bleeding gums, a toothache, problems eating or chewing food, or other problems in your mouth?

2) When was your last dental visit?

As part of routine counseling, health care providers should encourage all women to schedule a dental examination if it has been more than 6 months since their last examination or if they have any oral health problems. Patients often need reassurance that prevention, diagnosis, and treatment of oral conditions, including dental X-rays (with shielding of the abdomen and thyroid) and local anesthesia (lidocaine with or without epinephrine), are safe during pregnancy. Conditions that require immediate treatment, such as extractions, root canals, and restoration (amalgam or composite) of untreated caries, may be managed at any time during pregnancy. Delaying treatment may result in more complex problems. Counseling should include reinforcement of routine oral health maintenance, such as limiting sugary foods and drinks, brushing twice a day with fluoridated toothpaste, flossing once daily, and dental visits twice a year. Dental providers often recommend the use of chlorhexidine and fluoridated mouth rinses, and xylitol-containing chewing gum to decrease oral bacteria. No adverse effects have been reported with these products during pregnancy.

BIBLIOGRAPHY

1. Srinivas SK, Sammel MD, Stamilio DM, Clothier B, Jeffcoat MK, Parry S, et al. Periodontal disease and adverse pregnancy outcomes: Is there an association? Am J Obstet Gynecol. 2009;200:497.e1–8.

2. Boggess KA, Lieff S, Murtha AP, Moss K, Beck J, Offenbacher S. Maternal periodontal disease is associated with an increased risk for preeclampsia. Obstet Gynecol. 2003;101:227–31.

3. Cota LO, Guimarães AN, Costa JE, Lorentz TC, Costa FO. Association between maternal periodontitis and an increased risk of preeclampsia. J Periodontol. 2006;77:2063–9.

4. Ruma M, Boggess K, Moss K, Jared H, Murtha A, Beck J, Offenbacher S. Maternal periodontal disease, systemic inflammation and risk for preeclampsia. Am J Obstet Gynecol. 2008;198:389.e1–5.

5. Barak S, Oettinger-Barak O, Machtei EE, Sprecher H, Ohel G. Evidence of periopathogenic microorganisms in placentas of women with preeclampsia. J Periodontol. 2007;78:670–6.

6. Rustveld LO, Kelsey SF, Sharma R. Association between maternal infections and preeclampsia: A systematic review of epidemiologic studies. Matern Child Health J. 2008;12:223–42.

7. Contreras A, Herrera JA, Soto JE, Arce RM, Jaramillo A, Botero JE.

Periodontitis is associated with preeclampsia in pregnant women. J Periodontol. 2006;77:182–8.

8. Keelan JA, Wong PM, Bird PS, Mitchell MD. Innate inflammatory responses of human decidual cells to periodontopathic bacteria. Am J Obstet Gynecol. 2010:202.e1–471.

9. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. Int Dent J. 1975;25:229–35.

10. Offenbacher S, Jared HL, O'Reilly PG, Wells SR, Salvi GE, Lawrence HP, et al. Potential pathogenic mechanisms of periodontitis associated pregnancy complications. Ann Periodontol. 1998;3:233–50.

11. Lin D, Smith MA, Champagne C, Elter J, Beck J, Offenbacher S. Porphyromonas gingivalis infection during pregnancy increases maternal tumor necrosis factor alpha, suppresses maternal interleukin-10, and enhances fetal growth restriction and resorption in mice. Infect Immun. 2003;71:5156–62.

12. Boggess KA, Edelstein BL. Oral health in women during preconception and pregnancy: Implications for birth outcomes and infant oral health. Matern Child Health J. 2006;10(Suppl):S169–74.

13. Beck JD, Offenbacher S. The association between periodontal diseases and cardiovascular diseases: A state-of-the-science review. Ann Periodontol. 2001;6:9–15.

14. von Dadelszen P, Magee LA. Could an infectious trigger explain the differential maternal response to the shared placental pathology of preeclampsia and normotensive intrauterine growth restriction? Acta Obstet Gynecol Scand. 2002;81:642–8.

15. Golub LM, Payne JB, Reinhardt RA, Nieman G. Can systemic diseases co-induce (not just exacerbate) periodontitis? A hypothetical “two-hit” model. J Dent Res. 2006;85:102–5.

16. López NJ, Smith PC, Gutierrez J. Higher risk of preterm birth and low birth weight in women with periodontal disease. J Dent Res. 2002;81:58–63.

17. Kunnen A, Blaauw J, van Doormaal JJ, van Pampus MG, van der Schans CP, Aarnoudse JG, et al. Women with a recent history of early-onset pre-eclampsia have a worse periodontal condition. J Clin Periodontol. 2007

18. Shetty M, Shetty PK, Ramesh A, Thomas B, Prabhu S, Rao A. Periodontal disease in pregnancy is a risk factor for preeclampsia. Acta Obstet Gynecol Scand. 2010;89:718–21.

19. Moura da Silva G, Coutinho SB, Piscoya MD, Ximenes RA, Jamelli SR. Periodontitis as a risk factor for preeclampsia. J Periodontol. 2012;83:1388–96.

20. Siqueira FM, Cota LO, Costa JE, Haddad JP, Lana AM, Costa FO. Maternal periodontitis as a potential risk variable for preeclampsia: A case-control study. J Periodontol. 2008;79:207–15

ANNEXURE

ABBREVIATION

TNF – Tumor Necrosis Factor

IL - Interleukin

SNP - Single Nucleotide Polymorphism

TLR - Toll Like Receptor

sEng – soluble endoglin

VEGF - vascular endothelial growth factor

LPS – Lipopolysaccharide

PGE- Prostaglandin

CRP – C-Reactive protein

CAL – Clinical Attachment Level

PROFORMA

PROFORMA

NAME AGE: IP / OP NO:

ADDRESS:

SOCIOECONOMIC STATUS

OBSTETRIC CODE:

MARITAL HISTORY:

MENSTRUAL HISTORY:

HISTORY OF PRESENTING ILLNESS:

FAMILY HISTORY:

PAST HISTORY:

OBSTETRIC HISTORY: LMP:

EDD:

EXAMINATION:

PR; BP; RR;

OBSTRETIC EXAMINATION

INVESTIGATIONS

Urine R/E:

24 hr Urinary protein

Urine spot PCR

ROUTINE INVESTIGATIONS

DENTAL EXAMINATION: Clinical attachment loss

MASTER CHART

List of cases

Sl.No	Name	Age	Parity	Education	Socioeconomic class	Residence	Periodontitis	B.P	Proteinuria
1	Nithya	28	P1l1	High school	Class V	Rural	Present-mild	140/96	0.4
2	Helen	24	P2l2	Middle school	Class V	Rural	Present-moderate	150/92	0.8
3	Meghala	26	P1l1	High school	Class V	Rural	Absent	146/92	0.6
4	Nirmala	24	P1l1	Middle school	Class V	Rural	Present – mild	150/94	0.8
5	Rugmani	28	P2l2	High school	Class IV	Rural	Present – mild	160/100	0.6
6	Karthika	26	P1l1	High school	Class V	Rural	Present	150/92	0.5
7	Thilagam	25	P2l2	HSC	Class V	Urban	Present-severe	150/96	0.8
8	Deepa	26	P1l1	High school	Class V	Rural	Absent	140/96	0.6
9	Indra	24	P1l1	HSC	Class V	Rural	Present-mild	144/96	0.3
10	Rudra	26	P2l2	HSC	Class V	Rural	Absent	150/90	0.4

List of cases

Sl.no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
11	Vidhya	32	P3l3	High school	Class V	Rural	Present-moderate	150/92	0.8
12	Sandhya	24	P1l1	Middle school	Class V	Rural	Absent	140/94	0.3
13	Aiswarya	21	P1l1	Middle school	Class V	Rural	Present-mild	150/92	0.4
14	Anu	22	P2l2	HSC	Class V	Rural	Absent	148/94	0.7
15	Indhu	24	P1l1	High school	Class V	Rural	Present-mild	150/98	0.4
16	Janaki	32	P2l2	High school	Class V	Rural	Absent	144/96	0.3
17	Manohari	30	P2l2	Middle school	Class V	Rural	Present-mild	150/100	0.4
18	Deepthi	24	P1l1	HSC	Class V	Rural	Absent	160/100	0.4
19	Remya	32	P2l2	High school	Class V	Rural	Present-severe	150/98	0.5
20	Eswari	26	P1l1	High school	Class V	Rural	Absent	148/98	0.4

List of cases

Sl.no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
21	Kalyani	22	P1l1	High school	Class IV	Rural	present – mild	150/100	0.5
22	Radhika	24	P1l1	Middle school	Class V	Rural	Present-mild	146/98	0.6
23	Anitha	22	P1L1	HSC	Class V	Rural	Absent	150/90	0.4
24	Sruthy	24	P1l1	Middle school	Class V	Rural	Present	146/98	0.4
25	Rani	23	P1l1	High school	Class V	Rural	Absent	144/94	0.4
26	Nandini	19	P1l1	Middle school	Class V	Rural	Present-mild	150/100	0.7
27	Kousalya	23	P1l1	HSC	Class V	Urban	Present-mild	144/92	0.4
28	Keerthana	29	P1l1	Middle school	Class V	Rural	Present	146/98	0.5
29	Swathi	21	P1l1	HSC	Class V	Rural	Present – mild	150/96	0.8
30	Narmadha	21	P1l1	Middle	Class V	Rural	Present –	146/92	0.4

List of cases

				school			mild		
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontits	B.P	Proteinuria
31	Bhuvana	24	P2l2	HSC	Class V	Urban	Present moderate	150/96	0.4
32	Sundari	27	P2l2	Middle school	Class V	Rural	Present-moderate	146/90	0.5
33	Pushpa	21	P1l1	High school	Class III	Urban	Present-Severe	160/100	0.7
34	Anandhi	27	P2l2	Middle school	Class V	Rural	Absent	144/94	0.6
35	Kruthika	26	P1l1	HSC	Class V	Rural	present	150/100	0.4
36	Anbumani	27	P2l2	Midde school	Class V	Urban	Absent	144/96	0.5
37	Asha	28	P1l1	Graduate	Class III	Rural	Present – mild	150/94	0.6
38	Alamelu	22	P1l1	High school	Class V	Rural	Absent	140/98	0.4
39	Renuga	22	P2l2	HSC	Class V	Urban	Absent	144/92	0.4
40	Usha	25	P1l1	HSC	Class V	Rural	present	150/90	0.4

List of cases

Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
41	Bhavani	24	P2l2	HSC	Class V	Urban	Present moderate	150/96	0.4
42	Akshaya	26	P1l1	High school	Class V	Rural	Absent	144/96	0.6
43	Kundavi	24	P1l1	Middle school	Class V	Rural	Present –mild	146/98	0.5
44	Subha	21	P1l1	HSC	Class IV	Rural	Absent	150/104	0.6
45	Pawn	32	P3l3	Illiterate	Class V	Rural	Present severe	160/104	0.8
46	Anbu	28	P2l2	Middle school	Class V	Rural	Absent	144/92	0.4
47	Rukku	34	P3l3	Middle school	Class V	Rural	Absent	150/90	0.8
48	Pattamal	23	P1l1	Middle school	Class V	Rural	Present-mild	142/90	0.4
49	Ambiga	24	P1l1	HSC	Class V	Rural	Present moderate	150/96	0.4
50	Chithra	23	P1l1	HSC	Class V	Rural	Absent	148/92	0.7

List of cases

Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
51	Ambujam	23	P1l1	High school	Class V	Urban	Present-moderate	150/90	0.5
52	Revathy	22	P1l1	Middle school	Class V	Rural	Absent	144/94	0.6
53	Arathy	28	P2l2	High school	Class V	Urban	Present –mild	150/104	0.5
54	Suganya	31	P1l1	Middle school	Class V	Rural	Present mild	144/98	0.4
55	Bindhu	27	P1l1	HSC	Class V	Rural	Absent	150/90	0.5
56	Nandini	22	P2l2	Middle school	Class V	Rural	Absent	140/98	0.4
57	Dhanam	24	P2l2	HSC	Class V	Rural	Absent	150/90	0.5
58	Fathima	31	P2l2	High school	Class V	Rural	Absent	148/92	0.4
59	Gowri	25	P1l1	HSC	Class V	Rural	Present –mild	146/106	0.5
60	Hibina	28	P1l1	High school	Class V	Rural	Present –mild	150/98	0.7
61	Jaseema	22	P1l1	HSC	Class V	Rural	Absent	144/98	0.5
Sl no	Name	Age	Parity	Education	Socioeconomic	Residence	Periodontitis	B.P	Proteinuria

List of cases

					status				
62	Sainabha	22	P1l1	Illiterate	Class V	Rural	Present-mild	150/98	0.3
63	Sulthana	26	P2l2	HSC	Class V	Urban	Absent	144/98	0.4
64	Amritha	22	P1l1	High school	Class V	Rural	Present – moderate	150/90	0.5
65	Savithri	25	P2l2	HSC	Class V	Urban	Present-moderate	140/94	0.6
66	Brindha	29	P1l1	High school	Class V	Rural	Absent	160/90	0.4
67	Anamika	32	P2l2	graduate	Class III	Urban	Present-moderate	150/92	0.5
68	Reshmi	24	P1l1	HSC	Class V	Rural	Absent	140/92	0.4
69	Anjana	22	P1l1	High school	Class V	Rural	Absent	144/98	0.5
70	Vimala	26	P1l1	Middle school	Class V	Rural	Present-mild	150/94	0.5
71	Sundari	31	P2l2	Middle school	Class V	Rural	Absent	150/90	0.5
72	Babitha	28	P1l1	High school	Class V	Rural	Present –mild	144/96	0.4
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria

List of cases

73	Latha	22	P1l1	High school	Class V	Urban	Present-mild	150/90	0.4
74	Meena	28	P1l1	Middle school	Class V	Rural	Present –mild	150/94	0.6
75	Sreenidhi	21	P1l1	Middle school	Class V	Rural	Absent	144/92	0.4
76	Shankari	22	P1l1	Middle school	Class V	Rural	Absent	146/98	0.6
77	Umadevi	21	P1l1	HSC	Class V	Rural	Present-moderate	150/92	0.7
78	Nazreen	24	P1l1	HSC	Class V	Rural	Absent	144/94	0.5
79	Kavitha	22	P1l1	High school	Class V	Rural	Absent	146/98	0.4
80	Cathreine	24	Pl11	High school	Class V	Rural	Present-mild	150/92	0.5
81	Dona mary	26	P1l1	HSC	Class V	Rural	Absent	146/98	0.4
82	Hema	22	P1l1	HSC	Class V	Rural	Present-moderate	150/90	0.4
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
83	Veena	21	P1l1	High school	Class V	Urban	Absent	150/92	0.4

List of cases

84	Aseena	22	P1l1	HSC	Class V	Rural	Present-mild	144/96	0.5
85	Tulasi	21	P1l1	High school	Class V	Rural	Present-mild	160/92	0.7
86	Vidhya	22	P2l2	HSC	Class V	Rural	Absent	146/98	0.4
87	Priya	23	P1l1	High school	Class V	Urban	Present – moderate	150/92	0.5
88	Kamakshi	21	P1l1	High school	Class V	Rural	Absent	144/92	0.4
89	Kanaga	24	P1l1	High school	Class IV	Rural	Present-mild	150/92	0.6
90	Naveena	22	P1l1	HSC	Class V	Rural	Present-mild	144/96	0.4
91	Ivanjaline	23	P2l2	High school	Class V	Rural	Absent	150/98	0.5
92	Nirmala	25	P1l1	HSC	Class V	Urban	Present- mild	148/92	0.6
93	Pallavi	23	P1l1	Middle school	Class V	Rural	Present-mild	144/98	0.8
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
94	Raghavi	26	P1l1	High school	Class V	Rural	Present-mild	160/92	0.7
95	Seetha	24	P1l1	Middle school	Class V	Urban	Absent	144/98	0.4
96	Rakhi	19	P1l1	HSC	Class V	Rural	Present-mild	146/98	0.5
97	Nethra	20	P1l1	Middle	Class V	Rural	Present-mild	150/92	0.4

List of cases

				school					
98	Ann mary	31	P1l1	graduate	Class V	Rural	Present-severe	160/100	0.8
99	Firdose	32	P2ll2	Middle school	Class V	Rural	Present –mild	150/98	0.4
100	Laila	28	P1l1	High school	Class V	Rural	Present-mild	160/98	0.5

List of controls

Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
1	Krupa	22	P1l1	Middle school	Class V	Rural	Absent	120/80	0.2
2	Rasathi	24	P1l1	High school	Class V	Rural	Absent	122/82	0.1
3	Rejina	22	P1l1	HSC	Class V	Rural	Absent	110/70	0.1
4	Boomika	28	P1l1	High school	Class V	Urban	Present-mild	114/70	0.2
5	Malathy	22	P2l2	HSC	Class V	Rural	Absent	110/72	0.1
6	Punitha	23	P1l1	Middle school	Class V	Rural	Absent	120/74	0.2
7	Kala	25	P1l1	High school	Class V	Rural	Absent	110/72	0.1
8	Vanitha	29	P1l1	Middle school	Class V	Rural	Present-mild	120/70	0.2
9	Sudha	23	P1l1	HSC	Class V	Rural	Absent	100/70	0.2
10	Ammu	21	P1l1	High school	Class IV	Urban	Present-moderate	104/70	0.1
11	Sumna	26	P1l1	HSC	Class V	Rural	Absent	110/80	0.2

List of controls

Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
12	Rajitha	22	P1l1	HSC	Class V	Urban	Present – mild	110/70	0.2
13	Sumithra	28	P2l2	High school	Class V	Rural	Absent	104/60	0.3
14	Anu	22	P1l1	Middle school	Class IV	Rural	Absent	110/70	0.3
15	Rudra	26	P1l1	High school	Class V	Rural	Absent	100/60	0.2
16	Kannagi	21	P1l1	HSC	Class V	Rural	Present-mild	110/70	0.2
17	Selvi	22	P1l1	Middle school	classV	Rural	Absent	100/70	0.1
18	Renita	24	P1l1	High school	Class V	Rural	Absent	110/72	0.2
19	Ananya	26	P2l2	Middle school	Class V	Rural	Absent	100/70	0.1
20	Jothy	28	P1l1	High	Class V	Rural	Absent	120/70	0.2

List of controls

				scool					
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
21	Sathya	30	P2l2	Middle school	Class V	Urban	Present-mild	110/80	0.1
22	Neeraja	32	P2l2	High school	Class V	Rural	Absent	100/60	0.2
23	Arundadi	23	P1l1	Graduate	Class V	Rural	Absent	100/70	0.2
24	Suganthy	24	P1l1	Middle school	Class V	Rural	Present mild	110/70	0.2
25	Girija	22	P1l1	HSC	Class V	Rural	Absent	100/70	0.2
26	Ranjini	24	P1l1	High school	Class V	Rural	Present – mild	110/70	0.2
27	Evanjaline	22	P1l1	Middle school	Class V	Rural	Absent	100/60	0.3
28	Susan	24	P1l1	HSC	Class V	Rural	Absent	110/70	0.2
29	Tara	22	P1l1	Middle school	Class V	Rural	Present-mild	100/70	0.2
30	Priyanka	23	P1l1	HSC	Class V	Rural	Absent	110/70	0.1
31	Sudha	25	P1l1	Middle	Class V	Rural	Absent	104/70	0.2

List of controls

				school					
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
32	Bhavani	21	P1l1	High school	Class V	Urban	Absent	100/64	0.1
33	Supriya	22	P1l1	Middle school	Class V	Rural	Present-mild	100/80	0.2
34	Rajam	23	P1l1	HSC	Class V	Rural	Absent	110/80	0.1
35	Poorni	24	P1l1	Middle school	Class V	Rural	Absent	100/70	0.1
36	Soumya	21	P1l1	HSC	Class V	Rural	Absent	110/70	0.2
37	Sheeba	22	P2l2	High school	Class V	Rural	Present-mild	100/70	0.2
38	Stella	22	P2l2	Middle school	Class V	Rural	Absent	100/70	0.2
39	Monika	29	P2l2	High school	Class V	Rural	Absent	104/70	0.1
40	Monisha	30	P1l1	Middle school	Class V	Rural	Present – mild	110/70	0.2
41	Indra	31	P2l2	Middle	Class V	Rural	Absent	100/70	0.1

List of controls

				school					
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
42	Saranya	22	P1l1	Middle school	Class V	Rural	Absent	104/64	0.3
43	Karpagam	32	P3l3	High school	Class V	Rural	Present-mild	110/60	0.2
44	Vani	28	P1l1	High school	Class V	Rural	Absent	100/60	0.1
45	Midhula	21	P1l1	HSC	Class V	Rural	Absent	104/68	0.1
46	Sany rose	22	P1l1	Middle school	Class V	Rural	Absent	10/70	0.2
47	Jisha	29	P1l1	High school	Class V	Urban	Present-mild	104/70	0.1
48	Yamini	34	P1l1	Middle school	Class V	Rural	Present mild	100/64	0.3
49	Kanimozhi	28	P1l1	HSC	Class V	Rural	Absent	110/70	0.2
50	Vinaya	32	P2l2	Middle school	Class V	Rural	Absent	100/70	0.1
51	Reena	28	P1l1	High	Class V	Rural	Present-	104/72	0.2

List of controls

				school			mild		
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
52	Yalini	22	P1l1	High school	Class V	Urban	Absent	110/70	0.2
53	Saroja	21	P1l1	Middle school	Class V	Urban	Absent	104/60	0.1
54	Hepsibha	27	P1l1	High school	Class V	Rural	Absent	100/60	0.2
55	Zeenath	21	P1l1	Middle school	Class V	Rural	Present-mild	104/80	0.2
56	Aloka	23	P1l1	High school	Class V	Rural	Absent	110/70	0.4
57	Rajathy	29	P2l2	Middle school	Class V	Rural	Absent	104/72	0.3
58	Kalavathy	30	P2l2	High school	Class V	Rural	Present-moderate	110/70	0.2
59	Pankajam	32	P2l2	Middle school	Class V	Rural	Absent	110/70	0.3
60	Yasodha	24	P1l1	Middle	Class V	Rural	Absent	104/70	0.2

List of controls

				school					
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
61	Shankari	29	P1l1	Middle school	Class V	Rural	Absent	106/74	0.2
62	Paarvathy	28	P2l2	Middle school	Class V	Rural	Absent	110/72	0.2
63	Shivani	30	P1l1	High school	Class V	Rural	Absent	104/70	0.3
64	Pavithra	26	P1l1	HSC	Class V	Urban	Absent	100/70	0.2
65	Nimmi	23	P1l1	Middle school	Class V	Rural	Absent	120/80	0.4
66	Steffy	29	P1l1	HSC	Class V	Rural	Absent	110/80	0.2
67	Janet	30	P2l2	High school	Class V	Rural	Present-moderate	120/70	0.2
68	Sofia	29	P1l1	Graduate	Class V	Rural	Absent	110/70	0.3
69	Pitchamal	30	P2l2	Middle school	Class V	Rural	Absent	104/70	0.2
70	Roshni	31	P1l1	Middle school	Class V	Rural	Absent	100/60	0.2

List of controls

71	Sherin	30	P1l1	HSC	Class V	Rural	Absent	108/72	0.1
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
72	Merlin	32	P2l2	HSC	Class V	Rural	Absent	110/70	0.2
73	Sarasu	33	P1l1	Middle school	Class V	Rural	Present-mild	108/70	0.2
74	Kaveri	28	P1l1	HSC	Class V	Rural	Absent	104/70	0.1
75	Aradhana	23	P1l1	Middle school	Class V	Rural	Absent	110/70	0.2
76	Vardhini	27	P1l1	High school	Class V	Urban	Present-mild	100/70	0.3
77	Prathima	28	P1l1	Middle school	Class V	Rural	Absent	108/72	0.2
78	Benita	30	P2l2	Graduate	Class V	Rural	Absent	110/70	0.3
79	Divya	22	P1l1	Middle school	Class V	Rural	Absent	104/72	0.2
80	Christa	27	P1l1	High school	Class V	Rural	Absent	110/70	0.3
81	Elakiya	28	P1l1	Middle school	Class V	Urban	Absent	104/70	0.2

List of controls

83	Indhu	30	P1l1	HSC	Class V	Rural	Absent	110/70	0.3
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
84	Vimala	22	P1l1	Middle school	Class V	Rural	Absent	104/72	0.2
85	Udhaya	21	P1l1	Middle school	Class V	Rural	Absent	110/70	0.3
86	Sreeja	22	P1l1	Middle school	Class V	Rural	Present-moderate	104/72	0.2
87	Juliee	30	P2l2	HSC	Class V	Rural	Absent	110/70	0.3
88	Theju	28	P1l1	Middle school	Class V	Rural	Absent	104/70	0.2
89	ponni	29	P2l2	HSC	Class V	Rural	Present-moderate	110/70	0.3
90	Vanaja	30	P1l1	High school	Class V	Rural	Absent	104/70	0.2
91	Nila	28	P1l1	Middle school	Class V	Urban	Absent	108/72	0.2
92	Roopini	29	P1l1	High school	Class V	Rural	Absent	110/78	0.3

List of controls

93	Sharmila	28	P2l2	Graduate	Class V	Rural	Present-mild	102/64	0.2
Sl no	Name	Age	Parity	Education	Socioeconomic status	Residence	Periodontitis	B.P	Proteinuria
94	Smitha	22	P1l1	Middle school	Class IV	Urban	Absent	130/80	0.3
95	Sasirekha	22	P1l1	High school	Class V	Rural	Present-mild	110/70	0.2
96	Lavanya	32	P3l3	Middle school	Class V	Rural	Absent	108/72	0.2
97	Sambhavi	28	P2l2	High school	Class V	Rural	Absent	120/78	0.3
98	Prasanna	29	P1l1	Middle school	Class V	Urban	Absent	122/82	0.2
99	Nirmala	22	P1l1	High school	Class V	Rural	Absent	110/70	0.3
100	Sanuja	26	P2l2	HSC	Class V	Rural	Absent	120/80	0.2

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Suryakiranmayi.R.
II Year Post Graduate in MS (O&G)
Institute of Obstetrics & Gynaecology
Madras Medical College & RGGGH
Chennai 600 008

Dear Dr.Suryakiranmayi.R,

The Institutional Ethics Committee has considered your request and approved your study titled "**ASSOCIATION BETWEEN MATERNAL PERIODONTITIS AND PREECLAMPSIA ” - NO.05012017 (II).**

The following members of Ethics Committee were present in the meeting hold on **19.01.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.A.Rajendran,MS, Prof. of Surgery,MMC,Ch-3	: Member
6.Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch	: Member
7.Prof.Baby Vasumathi,MD.,Director, Inst. of O & G	: Member
8.Prof.K.Ramadevi,MD.,Director,Inst.of Bio-Che,MMC,Ch-3	: Member
9.Prof.R.Padmavathy, MD, Director,Inst.of Pathology,MMC,Ch-3	: Member
10.Prof.S.Mayilvahanan,MD,Director, Inst. of Int.Med,MMC, Ch-3	: Member
11.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

INFORMATION SHEET

We are selecting antenatal women according to the need for the study. We wish that you participate in this study.

- In this study, we will clinically check for periodonitis.The test you are subjected to, shall not affect you or your baby in uterus.
- Your participation in this study will not affect your AN care or any treatment if needed .
- The privacy of the patients in the research will be maintained throughout the study.
- In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in this study is voluntary. You are free to decide whether to participate in this study or withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the Investigator Signature of the Participant

PATIENT CONSENT FORM

Title: "ASSOCIATION OF MATERNAL PERIODONTITIS AND PREECLAMPSIA"

Name of the Investigator : **Dr. Suryakiranmayi.R**

Name of the Participant :

Name of the Institution : **IOG, Egmore, Chennai-8.**

I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in this study. I was free to ask any questions and they have been answered.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have informed the investigator of all the treatments I am taking or have taken in the past months/years including any native (alternative) treatments.
6. I have been advised about the risks associated with my participation in the study.*
7. I have not participated in any research study within the past ____ month(s). *
8. I am aware of the fact that I can opt out of the study at any time without having to give any reasoned this will not affect my future treatment in this hospital. *
9. I am also aware that the investigators may terminate my participation in the study at any time, for any reason, without my consent. *
10. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC if required.
11. I understand that my identity will be kept confidential if my data are publicly presented.
12. I have had my questions answered to my satisfaction.
13. I consent voluntarily to participate in the research/study.

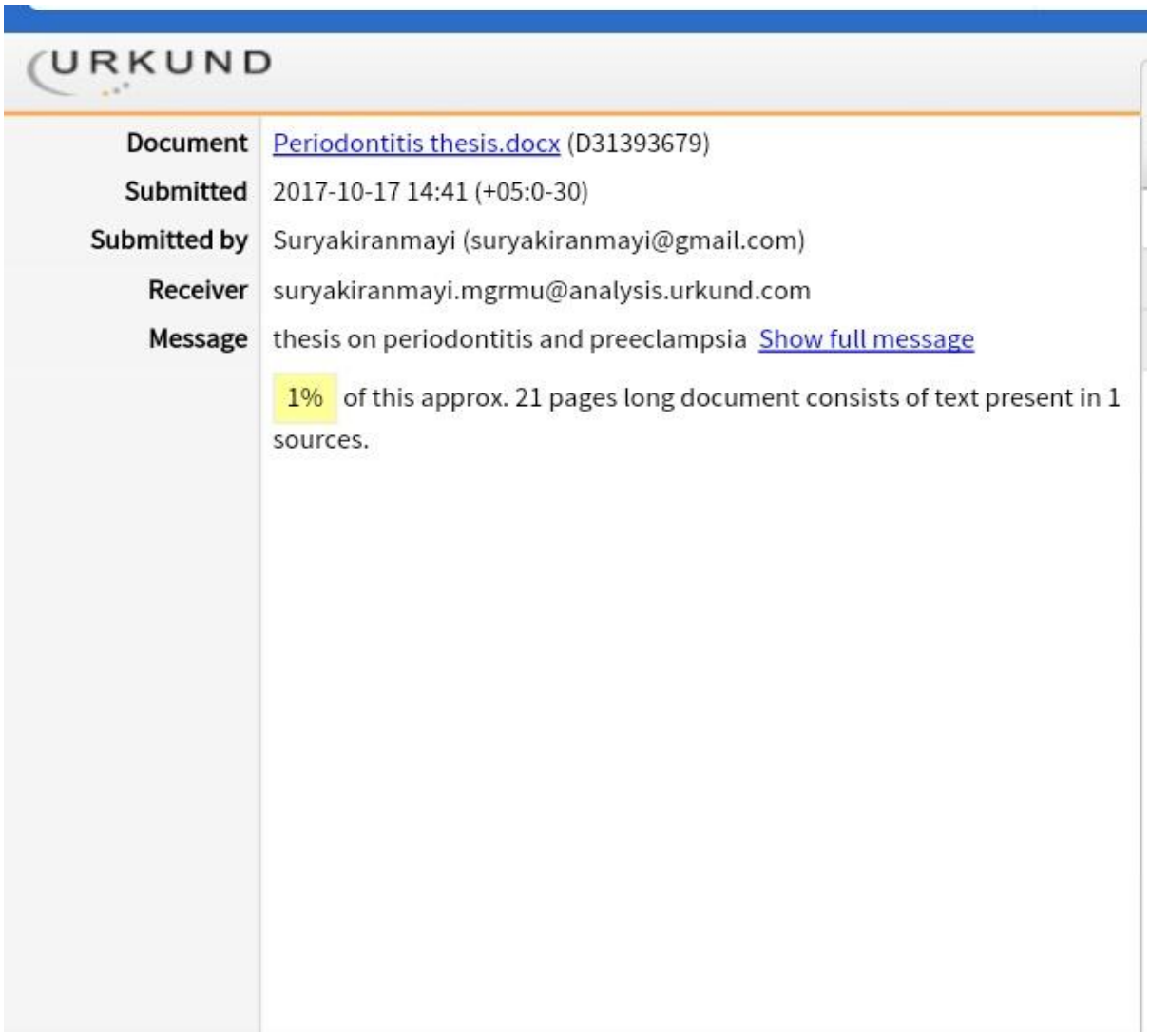
I am aware that if I have any question during this study, I should contact theinvestigator. By signing this consent form, I attest that the information given in this document has been clearly explained to me and understood by me. I will be given a copy of this consent document.

Signature/thumb imp of the patient

Date:

Place:

PLAGIARISM SCREENSHOT



PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled **"ASSOCIATION OF MATERNAL PERIODONTITIS AND PREECLAMPSIA"** of the candidate **Dr. SURYAKIRAMAYI.R** with registration number **221516015** for the award of **M.S** in the branch of **OBSTETRICS AND GYNAECOLOGY.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows **one percentage** of plagiarism in the dissertation (D31393679).

Signature and seal of guide